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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FORTY-EIGHTH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1926

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1926, to December, 1926, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

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DORNAN, PRINTER

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LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES[1]
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS[2]
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ
1913	JAMES CORNELIUS WILSON
1916	RICHARD H. HARTE
1919	WILLIAM J. TAYLOR
1922	THOMAS R. NEILSON
1925	HOBART A. HARE

[1] Died four months after his election.
[2] Resigned on account of ill-health.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

1926

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COMMITTEE ON NATHAN LEWIS HATFIELD PRIZE AND LECTURESHIP
(until February, 1928)

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(until February, 1929)

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FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1926

* Non resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., Professor of Hygiene and Bacteriology and Director of the School of Hygiene and Public Health, University of Pennsylvania. 4016 Pine St.

1921. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1926. Adler, Francis Heed, M.A., M.D., Assistant Surgeon, Wills Eye Hospital; Instructor in Ophthalmology and Physiology, University of Pennsylvania. 313 S. Seventeenth St.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Proctology in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1913. Alexander, Emory G., M.D., Surgeon to St. Christopher's Hospital for Children; Associate Surgeon to the Episcopal Hospital; Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania; Demonstrator of Fracture Dressings in the Jefferson Medical College, and Assistant Surgeon to the Kensington Hospital for Women. 337 S. Eighteenth St.

ELECTED

1906. Allen, Francis Olcott, Jr., A.B., M.D. 2216 Walnut St.

1896. Allyn, Herman B., A.B., M.D., Visiting Physician to the Philadelphia General Hospital and President of the Medical Board. 3910 Chestnut St.

1888. Anders, James M., M.D., LL.D., Professor of Medicine and Clinical Medicine in the Medico-Chirurgical College; Officer de l'Instruction Publique. 250 S. Seventeenth St.

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Gynecologist to the Stetson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1905. Appleman, Leighton F., M.D., Attending Surgeon to the Wills Eye Hospital; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School; Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant in Medicine and Students' Physician, University Hospital. 262 S. Twenty-first St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 257 S. Sixteenth St.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia General Hospital. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Professor of Research Medicine, University of Pennsylvania. John Herr Musser Department of Research Medicine, University of Pennsylvania.

1906. Babbitt, James A., A.B. (Yale), A.M. (Haverford), M.D., Assistant Professor of Otology, University of Pennsylvania; Associate Professor of Oto-Laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Laryn-

ELECTED

gologist and Aurist, Lankenau Hospital; Laryngologist, Mary Drexel Children's Hospital. 1912 Spruce St.

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Attending Surgeon to Wills Eye Hospital; Ophthalmologist, Howard Hospital. 1927 Spruce St.

†1892. Baker, George Fales, B.S., M.D. 1818 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

1889. Baldy, John Montgomery, M.D., Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and the Frederick Douglass Memorial Hospitals. Devon, Pa.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital; Demonstrator of Ophthalmic Surgery in the Philadelphia Polyclinic. 302 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1820 S. Rittenhouse Square.

1924. Bartle, Henry J., B.S., M.D., Clinical Assistant, Outpatient Medical Department, Lankenau Hospital; Clinical Assistant, Department for Diseases of the Stomach and Bowels, Jefferson Medical College and Hospital. 2018 Chestnut St.

1921. Bauer, Edward L., M.D., Professor of Children's Diseases, Jefferson Medical College; Pediatrist to the Jefferson and Memorial Hospitals and the Philadelphia Hospital for Contagious Diseases; Consulting Pediatrist, Germantown Hospital. 345 S. Nineteenth St.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1922. Bazett, Henry Cuthbert, M.A., M.D. (Oxon.), F.R.C.S. (England), Professor of Physiology, University of Pennsylvania. Bryn Mawr, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Associate Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 1919 Spruce St.

ELECTED

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy, Student Officer, U. S. Naval War College, Newport, R. I. 7 Cottage St., Newport, R. I.

1923. Bergey, David H., A.M., M.D., B.S., Dr. P.H., Professor of Hygiene and Bacteriology, University of Pennsylvania. 206 S. Fifty-third St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

1918. Billings, Arthur E., M.D., Clinical Assistant in Surgery, Dept. B, Jefferson Medical College; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 2020 Spruce St.

1917. Birdsall, Joseph C., A.B., A.M., M.D., Urologist, Polyclinic Section of the Medico-Chirurgical College and Hospital, Graduate School of Medicine, University of Pennsylvania; Assistant in Genito-urinary Dispensary, Presbyterian Hospital; Instructor in Surgery, University of Pennsylvania. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Professor of Obstetrics, Jefferson Medical College; Visiting Obstetrician, Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 1621 Spruce St.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital; Instructor in Gynecology in the University of Pennsylvania. Medical Arts Building.

1894. Bochroch, Max H., M.D., Neurologist to the St. Joseph's and Jewish Hospitals; Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

1923. Bond, Earl D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, Graduate School of Medicine, Univeristy of Pennsylvania. Bryn Mawr, Pa.

1911. Bonney, Charles W., A.B., M.D., Associate in Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1924. Boon, David J., M.D., Opthalmologist, Memorial Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Ophthalmic Surgeon, Episcopal Hospital. 1532 N. Fifteenth Street.

ELECTED

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Department, Jefferson Medical College; X-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 4910 Frankford Ave.

1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis, Graduate School, University of Pennsylvania; Physician to the Philadelphia General and Northwestern General Hospitals. 2024 Chestnut St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital. 82 W. Lacrosse Ave., Lansdowne, Pa.

1923. Bower, John O., M.D. 2008 Walnut St.

1891. Boyd, George M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Consultant to the Philadelphia Lying-in Charity; Consulting Obstetrician to the Preston Retreat; Obstetrician and Gynecologist to the Medico-Chirurgical Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Assistant Physican to the Orthopædic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1925. Boykin, Irvine M., M.D., Associate Surgeon to Episcopal Hospital; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. Elkins Park, Pa.

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Howard Hospital; Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

1921. Bransfield, John W., M.D., Clinical Professor of Surgery, Woman's Medical College; Visiting Surgeon to St. Agnes, St. Vincent's and Woman's College Hospitals. 2025 Walnut Street.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. Brinton, Lewis, M.D., Physician-in-Chief to the American Hospital for Diseases of the Stomach. 1301 Medical Arts Bldg., Sixteenth and Walnut Sts.

1900. Brinton, Ward, A.M., M.D., Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Visiting Physician, Department for Tuberculosis,

ELECTED

Philadelphia General Hospital, and Eaglesmere Sanatorium. 1423 Spruce St.

1917. Bromer, Ralph S., A.B. (Yale), M.D., Roentgenologist to the Episcopal and the Orthopædic Hospitals. 504 W. Mt. Airy Ave.

‡1919. Brown, H. MacVeagh, M.D., Associate Physician, Presbyterian Hospital. 4608 Baltimore Ave.

1919. Brown, Henry P., Jr., M.D., Surgeon to Pennsylvania and Children's Hospitals; Assistant Surgeon, Presbyterian Hospital. 624 Medical Arts Building.

1916. Brown, Samuel Horton, M.D. 1901 Mt. Vernon St.

1887. Brubaker, Albert P., A.M., M.D., Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Frankford, Phila.

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic and the College for Graduates in Medicine; Chief of the Dispensary for Diseases of the Ear at the University Hospital; Assistant Professor of Otology in the University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 2007 Chestnut St.

*1908. Cadbury, William W., A.M., M.D., Canton Christian College, Canton, China.

†1907. Cadwalader, Williams B., M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 1206 Medical Arts Building.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

ELECTED

1924. Carncross, Horace, M.D., 1003 Spruce St.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1926. Carpenter, Howard Childs, M.D., Vice-Dean for Pediatrics and Professor of Pediatric Hygiene, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania; Director, Department for the Prevention of Disease, Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Attending Ophthalmologist to the Bryn Mawr Hospital. 2030 Chestnut St.

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine Street.

1922. Case, Eugene A., M.D., Associate Professor of Pathology, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1892. Cattell, Henry W., A.M., M.D. 1806 Spruce St.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 315 S. Fifteenth St.

1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1926. Clarke, J. Alexander, A.B., M.D., Chief Clinical Assistant, Out-Patient Department of Applied Immunology, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital; Instructor in Medicine, Undergraduate School, University of Pennsylvania. 334 S. Twenty-first St.

ELECTED

1926. Clerf, Louis H., M.D., Instructor in Bronchoscopy and Esophagoscopy and Demonstrator of Laryngology, Jefferson Medical College; Assistant Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania; Chief Clinical Assistant in Bronchoscopy and Esophagoscopy, Jefferson Hospital; Assistant in Bronchoscopy and Esophagoscopy, University Hospital. The Esmond, Twelfth and Spruce Sts.

1910. Cloud, J. Howard, M.D., Attending Physician to the Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Ear, Throat and Nose, Pennsylvania Hospital; Consulting Laryngologist to the Philadelphia Orphanage and to the Sharon Hospital, Sharon, Conn. 1721 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1922. Cohen, Leon Solis, A.B., M.D., Clinical Assistant of Roentgenology, Jefferson Medical College. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor in Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence, Woman's Medical College of Pennsylvania; Director of the Jules E. Mastbaum Research Laboratory of the Jewish Hospital; Pediatrist to the Jewish Hospital. 1833 Pine St.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia General Hospital, to the Jewish Hospital and to the Rush Hospital. 1906 Walnut St.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Assistant Pediatrist, Polyclinic Hospital; Physician to Out-patient Department, Mary Drexel Home; Assistant Physician to Out-patient Department, Children's Hospital, University Hospital. Elkins Park, Pa.

1898. Coles, Stricker, M.D. Bryn Mawr, Pa.

*1901. Coley, Thomas Luther, A.B., M.D., Major, Medical Corps, U. S. Army. Fortress Monroe, Va.

ELECTED

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia General Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. Albany Apartments, Atlantic City, N. J.

1912. Copp, Owen, A.B., M.D., Consultant for the Future Development of the Pennsylvania Hospital. 4401 Market St.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. 1919 Cherry St.

1914. Corson, Edward Foulke, M.D., Assistant Professor of Dermatology, Jefferson Medical College; Dermatologist, Children's Hospital of Philadelphia. 1927 Spruce St.

1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

1924. Cowan, Alfred, M.D., Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Wills Hospital; Assistant Dispensary Surgeon, University Hospital; Assistant Instructor in Ophthalmology, School of Medicine, University of Pennsylvania. 2007 Pine Street.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. 2011 Pine Street.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Lecturer on Physiologic Optics in the Philadelphia Polyclinic and School for Graduates in Medicine; Ophthalmologist to the Philadelphia Orphanage. 1819 Walnut St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service, Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

ELECTED

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Instructor in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. 412 York Road, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., N. E. Cor. Sixteenth and Spruce Streets.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 2021 Pine Street.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth Street.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

ELECTED

1916. Davis, Warren B., M.D., Rhinologist, Laryngologist, and Otologist, St. Agnes' Hospital; Instructor in Surgery, Jefferson Medical College; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D. 4514 Springfield Ave.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 337 S. Eighteenth St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Visiting Surgeon, Hospital of the University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1830 DeLancey St.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1737 Chestnut St.

1923. De Long, Perce, M.D. 1909 Chestnut St.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Professor Emeritus of Nervous and Mental Diseases in the Jefferson Medical College; Foreign Corresponding Member of the Neurological Society of Paris, and Corresponding Member of the Psychiatric and Neurological Society of Vienna; Chevalier de l'Ordre National de la Légion d'Honneur. 1719 Walnut St.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 1807 Federal Reserve Bank Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth St.

1924. Dillon, Edward S., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Assistant Instructor in Medicine, School of Medicine, University of Pennsylvania. 2040 Pine St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 2002 Spruce Street.

ELECTED

1920. Doane, Joseph Chapman, M.D., Superintendent and Medical Director, Philadelphia General Hospital; Associate Professor of Internal Medicine, Graduate School of Medicine, University of Pennsylvania. Philadelphia General Hospital.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital. 25 East Washington Street, Chicago, Ill.

1907. Dorrance, George Morris, M.D., Professor of Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Professor of Surgery and Clinical Surgery, Woman's Medical College; Surgeon in Chief, Woman's College Hospital; Surgeon to St. Agnes' Hospital. 2025 Walnut St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Outpatient Department of the Pennsylvania Hospital; Physician to the Pennsylvania Institute for the Instruction of the Blind; Assistant Physician to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 1820 Pine Street.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1924. Egbert, Seneca, M.D., Professor of Hygiene, University of Pennsylvania. Wayne, Pa.

1911. Eliason, Eldridge E., B.A., M.D., Sc.D., Associate in Surgery in the University of Pennsylvania; Surgeon to the Howard Hospital; Assistant Surgeon to the University of Pennsylvania Hospital, the Philadelphia General Hospital and the American Stomach Hospital. 330 S. Sixteenth St.

1904. Elmer, Walter G., B.S., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Orthopædic

ELECTED

Surgery in the Woman's Medical College of Pennsylvania; Orthopædic Surgeon to the Polyclinic Hospital and to the Philadelphia General Hospital. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 3912 Chestnut St.

1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Medicine, Medical School, University of Wisconsin; Physician to State of Wisconsin General Hospital; Consulting Physician, Madison General and St. Mary's Hospitals. 1300 University Ave., Madison, Wis.

1905. Evans, William, M.D. 500 W. Gay St., West Chester.

1912. Eves, Curtis C., M.D., Otolaryngologist to Out-patient Department of the Pennsylvania Hospital; Attending Otolaryngologist to Bryn Mawr Hospital. 247 S. Seventeenth Street.

1894. Faries, Randolph, M.D. 2030 Chestnut St.

1922. Farley, David L., B.S., M.D., Instructor in Medicine, University of Pennsylvania; Assistant Physician, Pennsylvania Hospital. 1725 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 121 Maplewood Ave., Germantown.

1884. Fenton, Thomas H., M.D. 1319 Spruce St.

1907. Fetterolf, George, A.B., M.D., Sc.D., Professor of Otolaryngology, University of Pennsylvania. 2010 Spruce St.

1924. Fewell, Alexander G., M.D. 1924 Pine St.

1907. Fife, Charles A., A.B., M.D., Associate in Pediatrics in the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., M.D. Jenkintown, Pa.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes' and Pottstown Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

ELECTED

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital; Pathologist to the Children's Hospital. 3902 Locust St.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D. Overbrook, Pa.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 3600 Walnut St.

1916. Funk, Elmer Hendricks, M.D., Assistant Professor of Medicine in the Jefferson Medical College; Medical Director and Physician-in-Charge of the Department for Diseases of the Chest of Jefferson Hospital; Assistant Physician to the Pennsylvania Hospital; Visiting Physician to the White Haven Sanatorium. 1318 Spruce St.

1899. Gamble, Robert, G. M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D. S. E. Cor. Nineteenth and Spruce Sts.

*1913. Gates, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

‡1926. Geisler, Howard Dickes, A.M., M.D., Attending Physician, Germantown and Memorial Hospitals. 132 W. Walnut Lane, Germantown.

1917. Gerhard, Arthur Howell, M.D. 1615 N. Delaware Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899 Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. 1608 Spruce St.

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopædic Surgery, University of Pennsylvania; Surgeon to the Orthopædic Hospital; Orthopædic Surgeon to the Presbyterian Hospital; Chief Surgeon to the Widener Memorial Industrial Training School for Crippled Children. The Lenox, Thirteenth and Spruce Sts.

ELECTED

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D. Hamilton Court, Thirty-ninth and Chestnut Sts.

1906. Gittings, J. Claxton, M.D., Professor of Pediatrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital of Philadelphia. 1828 Pine St.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa.; Surgeon to the Dispensary of the Episcopal Hospital. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Medico-Chirurgical College. 2112 Pine St.

1906. Goepp, R. Max, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 1814 Spruce St.

1906. Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal and Misericordia Hospitals, Kensington Hospital for Women and Baltimore and Ohio Railroad. 2213 Walnut Street.

1908. Goodman, Edward H., M.D., Associate in Medicine in the University of Pennsylvania; Consultant to the Medical Dispensary of the University Hospital; Assistant Physician to the University Hospital; Assistant Physician to the Philadelphia General Hospital; Physician to the Presbyterian Hospital. Dorset, Vt.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1926. Grant, Francis C., A.B., M.D., Associate in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital. 133 S. Thirty-sixth St.

ELECTED

1904. Grayson, Charles P., M.D., Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of the Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital. 2213 Walnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty, University of Toronto School. 48 Bloor St., E., Toronto, Canada.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home and to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia General Hospital and to the Jefferson Medical College Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. N. E. Cor. Seventeenth and Walnut Sts.

1889. Hare, Hobart A., M.D., LL.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College. 1801 Spruce St.

ELECTED

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

1926. Hartman, Frederick L., A.B., M.D., Clinical Associate in Medicine, Lankenau Hospital; Assistant Instructor in Pathology, University of Pennsylvania; Assistant Pathologist, Visiting Staff of University and Philadelphia General Hospitals. 1914 Pine St.

*1888. Hartzell, Milton B., A.M., M.D., LL.D., Professor of Dermatology in the University of Pennsylvania. Fayetteville, Pa.

1907. Hatfield, Charles James, A.B (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1908. Heineberg, Alfred, P.D., M.D., Associate in Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes' Hospital. 1923 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 257 S. Sixteenth St.

1924. Hewson, William, A.B., M.D., Assistant in Otology, Graduate School of Medicine, University of Pennsylvania; Otolaryngologist, Pennsylvania Institution for Instruction of Blind and the Shriners' Hospital for Crippled Children; Assistant Otolaryngologist, Memorial Hospital. 6300 Overbrook Ave.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 5121 Pine St.

ELECTED

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 923 N. Sixty-fourth St.

*1892. Hinsdale, Guy, A.M., M.D. Hot Springs, Virginia.

*1888. Hirsh, A. Bern, M.D., Consulting Physician for Physiotherapy, Beth Israel Hospital. 67 W. Ninety-fourth St., New York City.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce St.

1924. Hirst, John C., 2nd, A.B., M.D., Instructor in Obstetrics, School of Medicine, University of Pennsylvania; Assistant Obstetrician, Philadelphia General and Howard Hospitals; Assistant Surgeon, Department of Obstetrics, University Hospital. 1918 Pine St.

*1908. Hitchens, Arthur Parker, M.D., M.C., U. S. A. Executive Offices, Malacanan, Manila, P.I.

‡1924. Hitschler, William A., M.D., Professor of Rhinology, Temple University. 1635 Spruce St.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian and the Germantown Hospitals; Associate Surgeon to the Pennsylvania Hospital; Associate Surgeon to the Widener Memorial Training School. 2019 Spruce St.

1913. Hoffman, Clarence, M.D., 1630 Pine St.

1906. Holloway, Thomas B., M.S., M.D., Sc.D. Professor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Orthopaedic Hospital and in Department of Neurology, Philadelphia General Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut Street.

1914. Hooker, Richard S., M.D. 2147 N. Howard St.

1919. Hopkins, Arthur H., B.S. (Haverford), M.D. 1726 Pine St.

1925. Hopkinson, R. Dale, M.D., Registrar and Assistant Physician, Abington Memorial Hospital. Jenkintown, Pa.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories in the University of Michigan. 1330 Hill St., Ann Arbor, Mich.

ELECTED

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D., Visiting Physician, Medical Department, Howard Hospital. N. E. Cor. Twentieth and Chestnut Sts.

1912. Hunter, John W., B.S., M.D. McDaniel, Md.

1921. Hunter, Robert J., M.D., Laryngologist, Philadelphia General Hospital; Chief of Dispensary and Assistant Laryngologist and Aurist, Howard Hospital; Instructor in Otology, University of Pennsylvania, Graduate School. 2011 Chestnut St.

1926. Husik, David N., M.D., Associate in Otolaryngology, University of Pennsylvania; Instructor in Otology, Graduate School of Medicine, University of Pennsylvania; Laryngologist and Otologist, Philadelphia General Hospital; Assistant Otolaryngologist, University Hospital. 1610 Spruce St.

1898. Hutchinson, James P., M.D., Surgeon to the Pennsylvania, the Methodist, the Children's, Memorial and the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. Penn Mutual Life Insurance Company, Sixth and Walnut Sts.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Plastic Surgeon to the Hospitals of the Graduate School of Medicine, University of Pennsylvania; Oral Surgeon, Episcopal Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D. C. 1503 Medical Arts Building.

1917. Jackson, Chevalier, M.D., Sc.D., Professor of Laryngology, Jefferson Medical College; Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania. 128 S. Tenth St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Secretary of State Commission for the Blind of Colorado. 217 Imperial Bldg., Denver, Colo.

1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Chester County Hospital. West Chester, Pa.

ELECTED

1922. Jameson, Howard Leon, M.D., Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Assistant Physician, Medico-Chirurgical and Polyclinic Hospitals. 2133 S. Sixteenth St.

1920. Janvier, G. Victor, M.D., Instructor in Obstetrics, University of Pennsylvania; Assistant Obstetrician to the Philadelphia General Hospital and the University of Pennsylvania Hospital; Associate in Gynecology at the American Stomach Hospital. Lansdowne, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. Rosemont, Pa.

1919. Jenks, Horace H., A.B., M.D., Medical Director, Associated Medical Clinics; Physician to the Out-Patient Department, Children's Hospital; Instructor in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 918 Clinton Street.

1926. Johnson, George Ernest, M.D. 5341 Chester Avenue.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1918. Jonas, Leon, M.D. 2253 N. Seventeenth St.

1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home. 256 S. Fifteenth St.

1924. Jones, Harold W., M.D., Demonstrator of Medicine, Jefferson Medical College; Assistant Physician, Philadelphia General Hospital. 1426 Spruce St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 103 S. Twenty-first St.

1900. Jopson, John H., M.D., Professor of Clinical Surgery, University of Pennsylvania; Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian and the Children's Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

1902. Jump, Henry D., M.D., Professor of Applied Therapeutics and Clinical Medicine, Woman's Medical College; Physician to Philadelphia General and Misericordia Hospitals. 2019 Walnut St.

ELECTED

1903. KALTEYER, FREDERICK J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. KARSNER, HOWARD T., M.D., Professor of Pathology in the Western Reserve University Medical School. 2109 Adelbert Rd., Cleveland, Ohio.

1926. KAUFMAN, ISADORE, M.D., Associate in Medicine, University of Pennsylvania; Medical Director, Kensington Dispensary for Tuberculosis; Associate Clinical and Sociological Director, Phipps Institute; Visiting Physician to White Haven Sanatorium. 316 Otis Building.

1923. KAY, JAMES, M.D., Visiting Physician, Episcopal Hospital; Assistant Instructor of Medicine, Jefferson Medical College. 600 W. Olney Ave.

1920. KEATING, PETER MCCALL, M.D., Chief of Surgical Dispensary, St. Agnes Hospital; Assistant Attending Surgeon, Bryn Mawr Hospital; Assistant, Surgical Out-patient Department, Pennsylvania Hospital; Pathologist, Chester County Hospital. Medical Arts Bldg., San Antonio, Texas.

1924. KEELER, J. CLARENCE, M.D., Associate Professor of Otology, Jefferson Medical College and Hospital; Assistant in Oto-laryngology, Germantown Hospital; Consultant Oto-laryngologist, Pottstown Hospital, Pa., and Newcombe Hospital, Vineland, N. J. Medical Arts Building.

†1867. KEEN, WILLIAM W., M.D., LL.D. (Hon.), Ph.D. (Brown, Upsala, etc.) (Hon.), Sc.D. (Jefferson and Harvard), Dr., Honoris Causa, University of Paris; (Hon.) F.R.C.S. (Eng., Edin. and Ireland). 1520 Spruce St.

1912. KEENE, FLOYD E., M.D., Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. Medical Arts Building.

1913. KELLY, FRANCIS JOSEPH, M.D. 2211 Locust St.

*1887. KELLY, HOWARD A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. KELLY, JAMES A., A.M., M.D., Visiting Surgeon to St. Mary's and Memorial Hospitals; Associate in Surgery in the Phila-

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delphia Polyclinic and College for Graduates in Medicine; Assisting Visiting Surgeon to St. Joseph's Hospital. 1900 S. Rittenhouse Square.

1912. Kelly, Thomas C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

1921. Kern, Richard A., A.B., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. 906 Medical Arts Building.

1920. Klauder, Joseph Victor, A.B., M.D., Professor of Dermatology, Woman's Medical College; Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Dermatologist, Misericordia, Woman's Medical College and American Stomach Hospitals; Assistant Dermatologist, Philadelphia General Hospital. 1934 Spruce Street.

1920. Klein, Thomas, M.D., Associate Professor of Medicine, Graduate School, University of Pennsylvania; Assistant Physician, Presbyterian Hospital. 250 S. Eighteenth St.

1913. Klopp, Edward J., M.D., Instructor in Surgery in the Jefferson Medical College; Assistant Surgeon to the Germantown Hospital; Chief Clinical Assistant in the Surgical Department of the Jefferson Hospital; Assistant Surgeon to the Out-patient Department of the Pennsylvania Hospital. 1611 Spruce St.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish and Mary J. Drexel Hospitals; Associate in Ophthalmology, Jefferson Medical College: Chief of Eye Clinic, Jefferson Hospital. Central Medical Bldg., Eighteenth and Chesnut Sts.

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Instructor in Obstetrics, University of Pennsylvania; Assistant Obstet-

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rician, University Hospital; Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 2018 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2022 Spruce St.

1925. Knox, Harry E., M.D., Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Germantown, St. Christopher's and Episcopal Hospitals; Surgeon to Philadelphia Hospital for Contagious Diseases. 719 Sixty-sixth Ave., Oak Lane, Phila.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., Professor of Pathology and Bacteriology, Graduate School of Medicine, University of Pennsylvania; Head of the Department of Pathology and Bacteriology in the Dermatological Research Laboratories of Philadelphia; Consulting Pathologist to St. Vincent's and Misericordia Hospitals; Consulting Serologist to Memorial and Germantown Hospitals. Cynwyd, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's Hospital for Children; Laryngologist to the Abington Hospital. 1701 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Director of Laboratories, Philadelphia General Hospital; Associate Professor of Pathology, Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. 127 N. Twentieth St.

*1909. Laird, J. Packard, M.D. 483 N. E. Twenty-seventh St., Miami, Florida.

1904. Landis, Henry R. M., M.D., Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. 1817 Pine St.

1907. Langdon, H. Maxwell, M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Ophthalmology, Under-Graduate School, University of Pennsylvania; Ophthal-

ELECTED

mologist of the Presbyterian, Orthopædic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 1332 Spruce St.

1926. Laws, George M., B.S., M.D., Associate Gynecologist, Presbyterian Hospital; Gynecologist, American Hospital for Diseases of the Stomach. 2033 Locust St.

‡‡1922. Leach, Wilmon W., M.D., Physician to Dispensary, Children's Hospital. 2118 Spruce St.

1920. Leavitt, Frederick Headley, M.D. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Assistant Professor, Epidemic Contagious Diseases, Graduate School of Medicine, University of Pennsylvania; Senior Pediatrist, Philadelphia Hospital for Contagious Diseases; Visiting Physician, Babies' Hospital, North Philadelphia. 2017 Spruce St.

1908. Lee, Walter Estell, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Germantown, Children's, Pennsylvania and Bryn Mawr Hospitals; Consulting Surgeon, Chester County Hospital, West Chester, Pa., and the Associated Medical Clinics. 905 Pine St.

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Honorary Professor of Chemistry, Wagner Free Institute of Science; Emeritus Pathological Chemist, Jefferson Hospital; Lecturer on Research, Philadelphia College of Pharmacy and Science. 1729 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1713 Walnut St.

1920. Leopold, Simon S., M.D., Instructor in Medicine, University of Pennsylvania; Assistant in Medicine, University Hospital. 2025 Spruce St.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 261 S. Seventeenth St.

1877. Lewis, Morris J., M.D., Attending Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and Emeritus Physician to the Pennsylvania Hospital. Ambler, Pa.

ELECTED

*1911. Lewis, Paul A., M.D. Princeton, N. J.

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Nervous Diseases, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Neurologist to the Philadelphia General Hospital and to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1893. Longaker, Daniel, M.D., Obstetrician to the Kensington Hospital for Women and Visiting Obstetrician to the Jewish Maternity Hospital. 1806 Spruce St.

1907. Loux, Hiram R., M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. Medical Arts Building, Sixteenth and Walnut Sts.

1921. Lucké, Baldwin, Dr. P.H., M.D., Assistant Pathologist, University and Philadelphia General Hospitals; Assistant Professor of Pathology, Medical School, University of Pennsylvania.

1924. Ludy, John B., A.B., A.M., M.D., Dermatologist, Lankenau and Methodist Hospitals; Assistant Dermatologist, Pennsylvania, Episcopal and Philadelphia General Hospitals. Medical Arts Building.

1923. Lueders, Charles W., A.B., M.D., Biochemist, Pennsylvania Hospital for Mental and Nervous Diseases; Research Worker, Gastro-enterology, Jefferson Medical College. Medical Arts Building.

1919. Lynch, Frank B., Jr., M.D., Dickson Fellow in Bacteriology to the Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Serologist, University Hospital; Instructor in Pathology, Evans Institute, University of Pennsylvania. 6028 Columbia Ave.

1914. Lyon, B. B. Vincent, A.B. (Williams Coll.), M.D. (Johns Hopkins), Associate in Medicine, Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department, Jefferson Hospital; Visiting Physician to Methodist Hospital. 328 S. Twenty-first St.

ELECTED

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology in the Medical Department of the University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 442 W. Stafford St., Germantown.

1905. McKenzie, R. Tait, M.D., LL.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine St.

*1916. McKnight, Howard A., A.B., M.D. Tecumseh Bldg., Springfield, Ohio.

1915. McLean, John D., M.D. 4316 Chestnut St.

1924. McMillan, Thomas M., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Cardiologist, Philadelphia General Hospital; Assistant Instructor in Medicine, School of Medicine, University of Pennsylvania; Instructor in Medicine, Graduate School of Medicine, University of Pennsylvania. 2044 Locust St.

1924. McPhedran, F. Maurice, A.B., M.D., Assistant in Clinical Research, Henry Phipps Institute. Seventh and Lombard Sts.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

1910. Mackinney, William H., M.D., Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant in the Urological Dispensary of the Lankenau Hospital. 1701 Chestnut St.

1901. MacLeod, George I., M.D. Ardmore, Pa.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Major, Charles P., B.S., M.D., Physician to the Dispensary of the Germantown Hospital; Pediatrist to the Dispensary of the Germantown Hospital; Pediatrist to the Abington Memorial Hospital. Tenth and Oak Lane.

ELECTED

1913. Manges, Willis F., M.D., Roentgenologist to the Jefferson Hospital; Director of the Roentgen-ray Laboratory in the Philadelphia General Hospital. 235 S. Fifteenth St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital. 1819 Spruce St.

1889. Martin, Edward, M.D., Professor of Surgical Physiology, University of Pennsylvania. 135 S. Eighteenth St.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1736 M. St., N.W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Assistant Surgeon to the Lankenau Hospital; Surgeon to the Out-patient Department of the Lankenau Hospital. 1816 Spruce St.

1923. Mendelson, Walter, M.D. 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Children's Hospital, Germantown Hospital, Children's Seashore House; Consulting Orthopædic Surgeon to the Howard Hospital and Glen Mills School. 2017 Spruce St.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. Miller, Edwin B., M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Associate in Ophthalmology, Temple University; Ophthalmologist, Roosevelt Hospital. 2008 Walnut St.

1921. Miller, T. Grier, A.B., M.D., Assistant Physician, University Hospital; Associate in Medicine, University of Pennsylvania. 110 S. Twentieth St.

1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 2121 DeLancey St.

*1917. Mitchell, A. Graeme, M.D., Professor of Pediatrics, College of Medicine, University of Cincinnati; Director of Pediatric and Contagious Services, Cincinnati General Hospital; Chief of Staff of Children's Hospital, Cincinnati. Cincinnati General Hospital.

ELECTED

1904. Mitchell, Charles F., M.D., Surgeon to the Pennsylvania, Bryn Mawr, and Germantown Hospitals. 2003 Pine St.

1921. Mohler, Henry K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

*1908. Montgomery, Charles M., A.B., M.D., St. Joseph's Sanatorium, Asheville, N. C.

1882. Montgomery, Edward E., A.M., M.D., LL.D., Emeritus Professor of Gynecology, Jefferson Medical College and Gynecologist to St. Joseph's Hospital, Retired. Veree Road, Fox Chase.

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Bronchoscopist to Philadelphia General Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

1924. Moorhead, Stirling W., A.B., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Assistant Surgeon, Department of Genito-urinary Surgery and in Urological Dispensary, University Hospital; Urologist, Howard Hospital. 1523 Pine St.

1918. Morgan, Arthur C., M.D., Professor of Applied Therapeutics, Medical Department, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Physician-in-Chief to the Frankford Hospital. 2018 Chestnut St.

1886. Morris, Caspar, M.D. 2050 Locust St.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital. 128 S. Eighteenth St.

1883. Morris, Henry, M.D., Emeritus Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Physician to St. Joseph's Hospital. 2310 Pine St.

1897. Morton, Samuel W., M.D. 2049 Chestnut St.

1905. Müller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals. 1930 Spruce St.

*1915. Musser, John H., Jr., B.S., M.D., Professor of Medicine and Head of Department, School of Medicine, Tulane University of Louisiana. 1551 Canal St., New Orleans, La.

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital. 1625 Spruce St.

*1896. Myers, T. D., M.D. 831 University Ave., Palo Alto, Calif.

ELECTED

1902. Nassau, Charles F., M.D., LL.D., Associate Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College. 1710 Locust St.

1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H., Narberth, Pa.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital; Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Attending Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. Radnor, Pa

1905. Norris, Charles C., M.D., Associate in Gynecology, University of Pennsylvania Medical School; Assistant Professor, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Hospital of the University of Pennsylvania; Gynecologist to the Children's Hospital, Philadelphia; Gynecologist and Obstetrician, Philadelphia General Hospital. Coronado, Twenty-second and Chestnut Streets.

1905. Norris, George William, A.B., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician-in-Chief, Medical Service "A", Pennsylvania Hospital; Attending Specialist (Heart and Lungs), U. S. Public Health Service (Veterans' Bureau). 321 S. Eleventh St.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Waverly Mills, S. C.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the

ELECTED

Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1924. Opie, Eugene L., A.B., M.D., Professor of Experimental Pathology, University of Pennsylvania; Director of Laboratories, Henry Phipps Institute. 2203 St. James Place.

1920. Ostheimer, Alfred J., A.B., M.D., L.R.C.P. (London), M.R.C.S. (England), Consulting Neurologist, State Hospital, Norristown, Pa. 6725 Ridge Ave.

1903. Ostheimer, Maurice, A.B., M.D. Whitford, Pa.

1913. Outerbridge, George W., A.B., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Professor of Clinical Surgery, Woman's Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Orthopaedic Hospital; Chief Surgeon, Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Chief of the Ear, Nose and Throat Service, Pennsylvania Hospital; Consulting Laryngologist and Aurist, Bryn Mawr Hospital; Consulting Aurist Chestnut Hill Hospital; Laryngologist, Children's Hospital of Philadelphia. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the Lankenau Hospital and Physician to the Medical Dispensary of the same; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the University of Pennsylvania and Roentgenologist to the University Hospital. Ardmore, Pa.

1909. Parish, Benjamin D., B.S., M.D., Associate Professor in Otology, Graduate School of Medicine, University of Pennsylvania; Aurist and Laryngologist to St. Agnes' and Chestnut Hill Hospitals. 1927 Spruce St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington

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Hospital for Women; Gynecologist to the Frankford Hospital. 1739 N. Seventeenth St.

1925. Patten, Clarence A., M.D., Associate in Neurology, Graduate School of Medicine, University of Pennsylvania; Assistant Visiting Physician, Orthopædic Hospital; Assistant Neurologist and Chief of Neurological Clinic, Polyclinic Hospital; Assistant Neurologist, Philadelphia General Hospital. 2037 DeLancey St.

1910. Patterson, Ross Vernet, M.D., Dean and Associate Professor of Medicine, Jefferson Medical College; Physician to the Philadelphia General Hospital; Physician to the Protestant Episcopal Hospital; Assistant Physician to the Jefferson Hospital. 2126 Spruce St.

1926. Paul, John Rodman, A.B., M.D., Director of the Ayer Clinical Laboratory; Associate in Surgical Pathology, Graduate School of Medicine, University of Pennsylvania; Fellow in Department of Research Medicine, University of Pennsylvania. 1941 Panama St.

1922. Pelouze, P. S., M.D., Associate in Urology, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. Medical Arts Building.

1909. Pemberton, Ralph, M.S., M.D., Physician to the Presbyterian Hospital; Director of the Department of Clinical Chemistry in the Pathological Laboratory of the Presbyterian Hospital. 2120 Sansom St.

1914. Pepper, O. H. Perry, B.S., M.D., Assistant Professor of Medicine, University of Pennsylvania. Medical Arts Bldg.

†1902. Pepper, William, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1917. Peter, Luther C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, University of Pennsylvania Graduate School. N. E. Cor. Twentieth and Chestnut Sts.

1912. Petty, Orlando H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Chief of the Department of Diseases of Metabolism, Hospitals of Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital. 1803 Pine St.

ELECTED

1905. Pfahler, George E., M.D., Assistant Radiologist, Medico-Chirurgical Hospital; Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Methodist and Abington Memorial Hospitals; Assistant Surgeon, Presbyterian and Lankenau Hospitals. 1822 Pine St.

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 1431 N. Fifteenth St.

1907. Phillips, Horace, M.D., Assistant Surgeon, U. S. Public Health Service; Examining Physician, Office of the District Attorney. 905 Land Title Building.

1911. Piersol, George Morris, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Philadelphia General and Methodist Episcopal Hospitals. 1913 Spruce Street.

1917. Piper, Edmund Brown, B.S., M.D., Associate in Obstetrics, University of Pennsylvania Medical School; Assistant Professor in Obstetrics, University of Pennsylvania Graduate School; Assistant Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital. 1936 Spruce St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. Pleasants, Henry, Jr., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopædic Hospital. West Chester, Pa.

1896. Posey, William Campbell, M.D., Ophthalmic Surgeon to the Howard Hospital; Consulting Ophthalmic Surgeon to the Wills Hospital; Chairman of the Commission on the Conservation of Vision for Pennsylvania. Radnor, Pa.

1899. Potts, Charles S., M.D., Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consulting Neurologist, Lankenau Hospital; Consulting Neurologist, Hospital for the Insane, Atlantic County, New Jersey, 2018 Chestnut St.

ELECTED

*1907. Price, George E., M.D. Suite 620, Paulsen Bldg., Spokane, Wash.

*1920. Quicksall, William E., M.D. E. 216 Fifteenth Ave., North, St. Petersburg, Fla.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; Attending Surgeon to the Wills Hospital. 1906 Chestnut St.

1913. Randall, Alexander, A.M., M.D., Assistant Professor of Surgery in charge of the Department of Urology, University of Pennsylvania. 1306 Medical Arts Building.

1887. Randall, B. Alex., M.A., M.D., Ph.D., Emeritus Professor of Otology in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb and to Memorial Hospital, Roxborough. 2009 Spruce St.

1926. Ravdin, I. S., M.D. 1930 Spruce St.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Hicks Ave., Columbia, Mo.

1925. Reeves, Rufus S., B.S., M.D., Associate in Medicine, Episcopal Hospital. 2227 Spruce St.

1920. Rehfuss, Martin E., M.D. N. E. Cor. 16th and Spruce Sts.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reimann, Stanley P., M.D., Director of the Research Institute, Lankenau Hospital; Assistant Professor of Experimental Pathology, Graduate School of Medicine, University of Pennsylvania. 516 Arbutus St., Germantown.

1923. Repplier, Sidney, J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. S. W. Cor. Fifteenth and Pine Sts.

‡1891. Rhoads, Edward G., M.D., New Hope, Pa.

*1910. Rhoads, Samuel, M.D. Altadena, Calif.

1919. Richardson, Russell, A.M., M.D., Director of the Laboratory, Methodist Episcopal Hospital; Pathologist to Abington Memorial Hospital and the Associated Medical Clinics. 320 S. Sixteenth St.

ELECTED

1919. Ridpath, Robert F., M.D., Professor of Rhino-laryngology, Temple University. 1737 Chestnut St.

1898. Riesman, David, M.D., Professor of Clinical Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish and Woman's Hospitals. 1520 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

1899. Roberts, Walter, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1732 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Episcopal, Samaritan and Garretson Hospitals. 327 S. Seventeenth St.

1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Professor of Surgery, Woman's Medical College; Surgeon-in Chief, Woman's College Hospital; Attending Surgeon, Bryn Mawr Hospital; Assistant Surgeon to the Presbyterian Hospital. 1310 Medical Arts Building.

1909. Rosenberger, Randle C., M.D., Professor of Preventive Medicine and Bacteriology in the Jefferson Medical College. 1001 Walnut St.

*1907. Royer, B. Franklin, M.D. 370 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., Clinical Professor of Orthopædic Surgery in the Woman's Medical College of Pennsylvania; Orthopædic Surgeon to the Jefferson Medical College; Orthopædic Surgeon to the Methodist and the Philadelphia General Hospitals. 1616 Spruce St.

1897. Sailer, Joseph, Ph.B., M.D., Professor of Clinical Medicine in University of Pennsylvania; Physician to the Philadelphia General, the University, and Presbyterian Hospitals. 250 S. Eighteenth St.

1900. Sajous, Charles E. de M., M.D., LL.D., Sc.D., Officer of the Legion of Honor of France and of the Order of Leopold of Belgium; Professor of Endocrinology, Graduate School of

ELECTED

Medicine, University of Pennsylvania; Emeritus Professor of Therapeutics in Temple University Medical School. 2043 Walnut St.

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the Lankenau Hospital. 1737 Chestnut St.

1906. Saylor, Edwin S., M.D., Chief Ophthalmologist to the Diagnostic Hospital of Philadelphia. 2100 Chestnut St.

1920. Scarlett, Hunter W., M.D. 230 S. Twenty-first St.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1917. Schnabel, Truman Gross, A.B. (Lehigh), M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, Philadelphia General Hospital; Physician in the Medical Out-patient Department, University Hospital. 1704 Pine Street.

1917. Schoff, Charles H., M.D. Media, Pa.

1921. Schumann, Edward Armin, A.B., M.D., Obstetrician and Gynecologist, Philadelphia General Hospital; Gynecologist, Frankford Hospital; Lecturer on Obstetrics, Jefferson Medical College; Consulting Gynecologist, Rush Hospital. 1814 Spruce St.

1887. de Schweinitz, George Edmund, A.M., M.D., LL.D., Professor of Ophthalmology in the University of Pennsylvania; Consulting Ophthalmic Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, The Philadelphia Polyclinic and School for Graduates in Medicine, and the Philadelphia General Hospital; Ophthalmic Surgeon to the University Hospital. 1705 Walnut St.

*1913. de Schweinitz, George Lord, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital;

ELECTED

Attending Surgeon to the Wills Hospital. 1417 N. Broad Street.

1917. Service, Charles A., M.D. Bala, Pa.

1924. Shallow, Thomas A., M.D., Assistant Surgeon, Jefferson Hospital; Demonstrator of Clinical Surgery, Jefferson Medical College; Surgeon to the Philadelphia General Hospital. 3942 Chestnut St.

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. N. E. Corner Seventeenth and Walnut Sts.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. Shoemaker, Harlan, A.B., M.D., Lecturer in Surgery, University of Southern California; Surgeon to Los Angeles County Hospital; Surgeon to Washington Street Clinic. 521 Bank of Italy Building, Los Angeles, Calif.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the Lankenau Hospital; Physician to the Out-patient Department of the Lankenau Hospital. 1727 Spruce St.

†1896. Shoemaker, William T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 109 S. Twentieth St.

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Lankenau, Children's and Oncologic Hospitals. 1737 Chestnut St.

1903. Sinclair, John Falconer, M.D., Epidemiologist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1226 Medical Arts Bldg.

ELECTED

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 2213 DeLancey St.

1920. Skillern, Ross Hall, M.D., Sc.D., Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist to Medico-Chirurgical Hospital. 1811 Spruce St.

‡1904. Smith, Allen J., A.M., M.D., Sc.D. (Penna. Coll.), LL.D. (McGill Univ.), Professor of Pathology and of Comparative Pathology and Director of Courses in Tropical Medicine in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Smith, S. MacCuen, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Memorial Hospital, Roxborough, and to the Oncologic Hospital. 1429 Spruce St.

1919. Smyth, Henry Field, M.D. Wayne, Pa.

1908. Speese, John, M.D., Associate Professor in Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals. 1832 Spruce St.

1895. Spellissy, Joseph M., A.M., M.D., Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopedic Department of the University Hospital. 317 S. Fifteenth St.

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 4409 Pine St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D. Consulting Surgeon to St. Agnes' Hospital. Haverford, Pa.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia at Wallingford and to the Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 1900 Rittenhouse Square.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital. 220 S. Sixteenth St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. 1728 Spruce St.

ELECTED

1901. Stevens, Arthur A., M.D., Professor of Materia Medica, Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes' Hospitals. 314 S. Sixteenth Street.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 1532 Pine St.

1925. Stokes, John H., A.B., M.D., Professor of Dermatology and Syphilology, School of Medicine, University of Pennsylvania; Professor of Dermatology, Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, University and Philadelphia General Hospitals; Dermatologist, Medico-Chirurgical and Polyclinic Hospitals. 3800 Chestnut St.

1921. Stoner, William H., A.M., Ph.C., Pharm. D., M.D., Associate Professor of Biochemistry and Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Assistant Metabolic Physician, Philadelphia General Hospital. Medical Laboratories, University of Pennsylvania.

1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society and the William Penn Charter School. 2015 Walnut St.

1923. Strecker, Edward, A., A.M., M.D., Professor of Nervous and Mental Diseases, Jefferson Medical College; Medical Director of Department for Mental and Nervous Diseases, Pennsylvania Hospital, and Director of the Neuro-psychiatric Clinic; Clinical Professor and Consultant in Psychiatry and Mental Hygiene, Yale University Medical School. 4401 Market St.

1923. Stroud, William D., B.S., M.D., Physician to the Pennsylvania Hospital and Chief of Out-Patient and Heart Clinics; Associate in Cardiology, Graduate School of Medicine, University of Pennsylvania; Instructor in Medicine, School of Medicine, University of Pennsylvania; Physician in Charge of the Children's Heart Hospital, Wynnefield, Pa. 1011 Clinton St.

1923. Strouse, Frederick M., M.D. 1301 Spruce St.

1884. Stryker, Samuel S., M.D., Consulting Physician to the Presbyterian Hospital. 3833 Walnut St.

*1900. Swan, John M., M.D., Attending Physician, Park Avenue Hospital. 457 Park Avenue, Rochester, New York.

ELECTED

‡1898. Sweet, William M., M.D., Professor of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Jefferson Medical College Hospital; Consulting Surgeon to the Wills Hospital. 1205 Spruce Street.

1900. Talley, James Ely, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian and Methodist Episcopal Hospitals. 1811 Spruce St.

1886. Taylor, John Madison, A.B. and A.M. (Princeton), M.D., Professor of Physical Therapeutics and Dietetics, Medical Department Temple University. 1504 Pine St.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. Taylor, William L., M.D. 2223 Tioga St.

†1910. Thomas, Benjamin A., A.B., A.M., M.D., Vice-Dean and Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Presbyterian Hospital; Consulting Genito-urinary Surgeon, Pennsylvania Railroad and to the Prison, Philadelphia. 1900 Spruce St.

1912. Thomas, Frank Wister, A.M., M.D., Visiting Physician to "Buttercup Cottage;" Consulting Physician to the Germantown Hospital. 27 E. Mt. Airy Ave.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 1737 Chestnut St.

1896. Thorington, James, A.M., M.D., Consulting Ophthalmic Surgeon to the Presbyterian Hospital; Ophthalmologist to the Ellwyn, Pa., Training School for Feeble-minded Children. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1912. Torrey, Robert G., M.D., Physician to the Philadelphia General Hospital; Associate in Medicine, University of Pennsylvania. 1716 Locust St.

1896. Toulmin, Harry, M.D., Vice-President, Medical Department, Penn Mutual Life Insurance Company. Haverford, Pa.

ELECTED

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1737 Chestnut St.

1926. Tucker, Gabriel, M.D., Cynwyd, Pa.

*1901. Tucker, Henry, M.D. Bellevue, Md.

†1894. Tunis, Joseph Price, M.D., Assistant Professor of Histology and Embryology, Temple University. Chestnut Hill, Philadelphia.

1924. Turner, Charles Root, A.B., D.D.S., M.D., Dean, School of Dentistry, University of Pennsylvania. Merion, Pa.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. 1731 Pine St.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia General Hospital; Physician to the Rush Hospital, the Philadelphia Lying-in-Charity Hospital and the Children's Aid Society of Philadelphia. 1506 Spruce St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Blairstown, N. J.

°1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. 1831 Chestnut St.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

1893. Vansant, Eugene Larue, M.D. 1737 Chestnut St.

1912. Vaux, Norris Wistar, M.D., Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 1810 S. Rittenhouse Square.

*1897. Veasey, Clarence A., A.M., M.D. Suite 404, Paulsen Building, Spokane, Wash.

†1883. Vinton, Charles Harrod, A.M., M.D. P. O. Box 464, Atlantic City, N. J.

ELECTED

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-patient Department of the Pennsylvania Hospital. 1814 Pine St.

1907. Walker, Warren, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium. Medical Arts Building.

1910. Ward, E. Tillson, A.M., M.D. 2006 Mt. Vernon St.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

1923. Watson, William R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Chief Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1903. Weber, Charles H., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania; Physician to the Medical Dispensary, Children's Hospital; Pediatrist, Howard Hospital and to the Municipal Court. 1721 Pine St.

1920. Weidman, Fred. D., M.D., Professor of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden; Attending Dermatologist, Philadelphia General Hospital. 20 Tenby Road, Llanerch, Pa.

1906. Weisenburg, Theodore H., M.D., Professor of Neuro-Psychiatry, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital and Physician to Orthopædic Hospital and Infirmary for Nervous Diseases. 2037 DeLancey St.

1923. Weiss, Edward, M.D., Physician to the Tuberculosis Wards, Philadelphia General Hospital; Associate in Pathology, Jefferson Medical College. 1923 Spruce St.

1913. Wells, P. Frailey, A.M., M.D., Ph.D., 754 N. Fortieth St.

1893. Westcott, Thompson S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Pediatrist to the Methodist Episcopal Hospital; Consulting Physician, Haddock Memorial Home. 1720 Pine St.

1921. Whitaker, William, M.D., Surgeon to Dispensary of the Episcopal Hospital. 5448 Germantown Ave.

ELECTED

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia. 6611 N. Tenth St.

*1905. Whiteway, Harold M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. Whiting, Albert D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 333 S. Eighteenth St.

1925. Widmann, Bernard P., M.D., 1321 Spruce St.

1914. Willard, de Forest P., B.S. (Univ. of Penna.), M.D., Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Polyclinic and Delaware Hospitals and Shriners' Hospital for Crippled Children. 1729 Spruce St.

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1924. Williams, Horace James, M.D. 5908 Greene St. Germantown.

1920. Williams, Philip F., Ph.B., M.D., Assistant Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Instructor in Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the Maternity Hospital of Philadelphia and Assistant Gynecologist to the Presbyterian Hospital. 2206 Locust St.

1916. Wilmer, Harry B., M.D., Assistant Instructor in Medicine, University of Pennsylvania: Assistant Visiting Physician and Assistant Neurologist to the Germantown Hospital; Visiting Chief to the Dispensary of the Germantown Hospital. 138 W. Walnut Lane, Germantown.

1921. Wilson, George, M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Clinical Professor of Neurology, Women's Medical College of Pennsylvania; Neurologist, Philadelphia General, Episcopal and Abington Memorial Hospitals; Assistant Neurologist, University Hospital. N. E. Cor. Twentieth and Chestnut Sts.

1874. Wilson, James Cornelius, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of

ELECTED

Clinical Medicine in the Jefferson Medical College; Physician-in-Chief (Emeritus) to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 2018 Chestnut St.

1897. Wilson, W. Reynolds, M.D. Villa Nova, Pa.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. 21 North Grand Ave., Baldwin, N. Y.

1918. Wolferth, Charles Christian, A.B., M.D., Associate in Medicine, University of Pennsylvania; Associate in Cardiac Pathology, William Pepper Laboratory of Clinical Medicine, University of Pennsylvania. 1704 Pine St.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's and the Howard Hospitals. 2035 Walnut St.

1900. Wood, George, B., M.D., Laryngologist and Otologist, Orthopædic and Howard Hospitals; Professor of Laryngology, Graduate School, University of Pennsylvania. N. E. Cor. Twentieth and Chestnut Sts.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Philadelphia College of Pharmacy and Science. 319 S. Forty-first St.

°1880. Woodbury, Frank, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 101 Allen Lane, Mt. Airy.

*1911. Woods, Andrew H., A.B., M.D., Professor and Head of the Neurological Department, Peking Union Medical College, Peking, China.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. Worden, Charles B., A.M., M.D. Princeton, N. J.

1894. Zentmayer, William, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital; Ophthalmologist to the Glen Mills School. 1506 Spruce St.

ELECTED

‡1899. Ziegler, S. Lewis, A.M., M.D., LL.D., Attending Surgeon to the Wills Hospital; Chief Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophthalmologie. 1625 Walnut St.

1887. Ziegler, Walter M. L., A.M., M.D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1911. Abbe, Robert, M.D. 40 W. Fifty-ninth St., New York City, New York.

1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1880. Carrow, Flemming, M.D., 823 David Whitney Building, Detroit, Mich.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1892. Emmet, Thomas Addis, M.D., 91 Madison Avenue, New York City, New York.

1916. Estes, William Lawrence, M.D., South Bethlehem, Pa.

1912. McCaw, Brig.-Gen. Walter D., M.D. Assistant Surgeon-General, U. S. A., Army Medical School, Washington, D. C.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.

1906. Pilcher, Lewis Stephen, M.D., 145 Gates Avenue, Brooklyn, New York.

1906. Shattuck, Frederick C., M.D., 135 Marlborough Street, Boston, Massachusetts.

1894. Warren, J. Collins, M.D., 58 Beacon Street, Boston, Massachusetts.

1894. Weir, Robert F., M.D., 16 East Ninety-sixth St., New York City, New York.

1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1890. Baccelli, Guido, Rome, Italy.
1924. Hopkins, Sir F. Gowland, M.D., Cambridge, England.
1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., Department of Biochemistry, McGill University, Montreal, Canada.
1922. McCarrison, Lieut.-Col., Robert, M.D., C.I.E., D.Sc. LL.D., F.R.C.P. (London), Pasteur Institute, Coonoor, S. India.
1924. Macleod, John James R., M.D., Toronto, Canada.
1906. Myles, Sir Thomas, M.D., 33 Merion Square, W., Dublin, Ireland.
1885. Rendu, Jean, M.D., Lyons, France.
1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., Ross Institute and Hospital for Tropical Diseases, Putney Heath, London, S.W. 15, England.

NECROLOGICAL LIST

FELLOWS

S. LEWIS ZIEGLER, M.D.	January 4, 1926
EDWARD G. RHOADS, M.D.	January 24, 1926
ALLEN J. SMITH, M.D.	August 18, 1926
WILLIAM J. HITSCHLER, M.D.	September 1, 1926
WILMON W. LEACH, M.D.	September 26, 1926
H. McVEAGH BROWN, M.D.	October 4, 1926
HOWARD DICKES GEISLER, M.D.	November 12, 1926
WILLIAM M. SWEET, M.D.	December 24, 1926

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

NOTE—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York.
1923	Edward P. Heller, M.D.	Kansas City, Mo.
1924	Gordon Cameron, M.B., B.S.	Victoria, Australia.
1925	Raphael Isaacs, M.D.	Boston.
1926	P. S. Pelouze, M.D., and Frederick S. Schofield, M.D.	Philadelphia.

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.
1924	George J. Heuer, M.D.	Cincinnati.
1926	Knud Faber, M.D.	Copenhagen.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N.Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D. and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Macleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).
Mar. 5, 1924	Prof. A Biedl	Prague (Czecho-Slovakia).
April 15, 1924	J. G. FitzGerald, M.D.	Toronto.
Oct. 31, 1924	John I. Hunter, M.D., and N. D. Royle, M.D.	Sydney (Australia).
Dec. 4, 1924	Willem Einthoven, M.D.	Leyden (Holland).
April 21, 1925	A. N. Richards, A.M., Ph.D.	Philadelphia.

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.
1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forrest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M. D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.

1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D,	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Prof. H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.
1924	Evarts Graham, M.D.	St. Louis.
1925	Edward Archibald, M.D.	Montreal, Can.
1926	Walton Martin, M.D.	Ithaca, N. Y.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D,	New York.
1925	Simon Flexner, M.D.	New York.

MEMOIR OF RICHARD H. HARTE, M.D.*

By GEORGE W. NORRIS, M.D.

On November 14, 1925, far from home but surrounded by his children and upon the banks of the beloved Mississippi, died Richard H. Harte.

Thus ended a life of seventy years—a life containing such joy and sorrow, such hope and despondency, such peace and strife as is the common lot of humanity. But the keynote of this human life which we are here to recall to memory, was service.

Born in 1855, at Little Rock, Illinois, descendant of a distinguished Irish family, which had been actively identified with Trinity College, Dublin—among whom one finds a clergyman, a noted traveler and a celebrated mathematician—Richard Harte was early left fatherless.

His father, William H. Harte, erstwhile officer in the Royal Navy, had settled in Illinois and become owner and captain of a well-known Mississippi steamboat. Receiving a commission in our Army soon after the outbreak of the Civil War, he saw much active service under Commodore Foote and Admiral Porter. His gallant career was tragically ended by an explosion on the *Mound City* while in action, and those aboard of her were scalded to death, drowned or shot while attempting to reach the shore.

It was for the purpose of inspecting a marker which he had had erected to commemorate this event that Dr. Harte undertook his last journey to the great river.

The details of Dr. Harte's career will be appended. Let us now but touch upon a few phases of his life that will perhaps reveal the man.

His early career, begun without financial resources, through the aid of his father's Philadelphia friends, illustrates the confidence which his personality inspired. That he gained and held the fervent

* Read May 10, 1926.

affection of these friends through life shows that their kindness was not misplaced.

That he married a fascinating and highly intellectual lady and became the father of worthy children, who lived to do him credit, betokens his personal charm and the high character of his intimate private life.

That he was revered and loved by a host of patients who lived because of his skill and blessed him because of his kind and gentle ways, reveals his innate sympathy.

The fact that his mother died following an emergency operation which he himself was forced to perform; that his wife died while he was serving his country in France; that his best friend died almost at his prime and at the threshold of success, shows that he knew sorrow and could meet it with fortitude.

His long connection with the University of Pennsylvania—from intern to adjunct professor of surgery—bespeaks an able worker. His many years of service in connection with three of our best hospitals reveals his conscientiousness and surgical skill. His faithful service as manager of the Episcopal Hospital evidences interest in institutions and their proper functionation.

His election as councilman from the eighth ward, and later his appointment as director of the Department of Public Health and Charities of this city—during which latter he planned and, but for the political squabbles of others, would have actually begun the new Philadelphia General Hospital, depicts his interest in civic matters and his willingness to serve.

His presidency of the American Surgical Association, of the College of Physicians of Philadelphia and of the Philadelphia Academy of Surgery reveals the measure of his professional achievements and his standing among his confrères.

His work at the American Ambulance in Paris, in 1916, his activities in connection with the American Red Cross, his organization and command of Base Hospital No. 10 in France during the World War, bespeak his patriotism.

The fact that despite his sixty-two years he served for weeks, midst frequent bombing by aviators, as head of a surgical emergency team on the Belgian front, sent hurriedly forward to help in stemming the German advance, betokens the sturdy stuff of which he was made.

Surely the receipt of the Order of St. Michael and St. George

from the British Government, of the order of Leopold from the Belgian Government, of a Fellowship in the Royal College of Surgeons of Ireland and of our own Distinguished Service Medal is more than a fortuitous coincidence.

But all these honors he valued less than his friendships—by their companions ye shall know them—and, if I judge rightly, his intimate friends, who were known more for their integrity than their numbers, valued Richard Harte far more for the man that he was than all else. He was not what the venacular of the day describes as a "good mixer." *Hoi polloi* made as little appeal to him as he to them.

He was a man of fearless truth. Time and time again we have seen him rise in council, reluctantly rise, when others failed to do so, to blight an unworthy candidate or an unjust cause. Anglo-Saxon in appearance, Keltic in temperament, his blue eyes seemed to grow bluer in moments of joy or anger and to intensify the blondness of his hair. He was a staunch friend and an implacable enemy.

His early life was a struggle against ill health and poverty, a combination which doubtless accounted for an unfavorable impression which he sometimes gave to strangers. During his later days neither of these factors obtained and his manner mellowed much.

Faultless he was not. He chafed under restraint, was forgetful of minor details often to a point of exasperation, was known to act impulsively when stimulated by what at the moment appeared to be a just cause; was positive in his opinions, often intolerant in his views and sometimes confused his principles with his prejudices. Yet nothing, perhaps, bespeaks his sterling merit more than that his very faults endeared him most to those who knew him best. "For though thy faults were thick as dust in vacant chambers, yet could I trust—thy kindness."

Two clarion notes of his character were sentiment and loyalty. Recall the reason of his last pilgrimage to the Mississippi. He felt that real kindness received could never be repaid. Recall his loyalty to those who had shown him kindness; his loyalty to his forbears, his teachers, his friends, his country and to the institutions which he held dear. He despised disloyalty, or, perhaps it might be more truthfully said, he could not understand it.

There was an imponderable and intangible value about Richard H. Harte which cannot be assayed or even described: a value which far exceeded his manifold achievements and obvious accom-

plishments. It far transcended his skill, his lovableness, his energy, his determination, his loyalty and the other fine qualities which he possessed. It was he himself—Harte—the personality—the man!

Richard Hickman Harte, the son of William Harte and Mary A. Betty, was born near Little Rock, Illinois, October 23, 1855. He received his early education in local schools, and later, under tutors, was prepared for the University of Pennsylvania, from which he was graduated in 1878. After a competitive examination he was elected intern in the University Hospital, and subsequently spent some time in the hospitals of Europe.

On returning to Philadelphia, Dr. Harte was appointed intern at the Pennsylvania Hospital in this city, and served the usual term of eighteen months. He later became assistant demonstrator of anatomy and surgery in the University of Pennsylvania and appointed surgeon to the University Hospital Dispensary. He later filled the following positions:

1883. Attending surgeon, Surgical Out-patient Department, Pennsylvania Hospital. Assistant in the Medical School University of Pennsylvania to Dr. D. Hayes Agnew and to Dr. John Ashhurst. Demonstrator of osteology.

1889. Surgeon to St. Mary's Hospital.

1891. Surgeon to the Episcopal Hospital. Surgeon to the Orthopedic Hospital.

1893. Surgeon to the Pennsylvania Hospital. Later consulting surgeon to St. Timothy's, Bryn Mawr and Abington Hospitals.

The professional organizations in which Dr. Harte was enrolled include the College of Physicians and the Philadelphia Academy of Surgery, in both of which he held fellowships, serving as treasurer of the former, and later as president of each organization. He was a member of the American Society of Clinical Surgery and of the American Surgical Association, having served as president of the latter and also as its recorder and editor. For many years he edited the University Medical Magazine.

He was a member of the Academy of Natural Sciences and the Zoölogical Society of Philadelphia, serving on the board of directors of the latter society from 1915 until the time of his death. He was a member of the board of managers of the Episcopal Hospital for many years and served on the executive committee, and from 1920 until the time of his death was secretary of the board.

Dr. Harte found time to also take an interest in civic affairs. In 1907, during the reform movement in Philadelphia, he represented the eighth ward in select council and was at one time director of the Department of Health and Charities. He developed plans for a reconstruction of Blockley, at a proposed cost of $6,000,000.

After leaving public office, Dr. Harte was active in aiding in the raising of relief funds for Belgium. He was influentially identified with the movement to procure a portion of Mount Desert Island, Maine, for a public reservation, so that certain mountains and watersheds might never be encroached upon for habitation but the natural beauty of the landscape forever preserved.

Before the United States entered the World War, Dr. Harte served in France as one of the surgeons at the American Ambulance Hospital at Neuilly, where he specialized in facial reconstruction work, performing many delicate operations. By appointment of President Wilson he served on a medical committee to standardize first-aid appliances.

He organized and commanded Base Hospital No. 10, recruited from the Pennsylvania Hospital, sailing for France May, 1917, and later became commanding officer of No. 16 General Hospital, B. E. F., at Treport, where nearly 43,000 wounded British were cared for. Despite the fact that he was sixty-two years of age at the time, he served for many weeks as head of an emergency surgical team, during one of the big German "drives," at a British Casualty Clearing Station (equivalent to our Field Hospital) on the Belgian front, near Poperinghe.

In recognition of his war services, Dr. Harte was awarded the Order of St. Michael and St. George by the British, the Order of Leopold by the Belgian and the Distinguished Service Medal by the United States governments. He was also elected, with five other American surgeons, a Fellow of the Royal College of Surgeons of Ireland. At the termination of the war he ranked a Colonel in the Medical Corps U. S. Army, was honorably discharged on January 31, 1919, and reaccepted a colonelcy in the Medical Reserve Corps at that time.

September 12, 1888, Dr. Harte married Maria H. Ames, daughter of Oakes A. Ames, of Boston, who died March 13, 1918. They had three children: Katharine, Mrs. George Putnam, of Boston; Richard, of Boston; and Helen, Mrs. R. Ellison Thompson, of Philadelphia.

Dr. Harte was a member of the Philadelphia, the Rittenhouse and the Corinthian Yacht clubs in Philadelphia, and of the Century Club in New York.

Dr. Harte left Philadelphia Monday, November 2, 1925, to join Dr. William Mayo on his boat at Memphis. He was taken ill with pneumonia on his arrival there and died at the Vicksburg Sanitarium on November 14. He was buried at the Village Cemetery, North Easton, Mass.

MEMOIR OF LOUIS STARR, M.D., LL.D.*

By THOMPSON S. WESTCOTT, M.D.

Louis Starr, for fifty years a Fellow of this College, a member at various times of its Council, and in later years a Censor, died after a short, acute illness at his home in Dinard, France, on the coast of Brittany, September 12, 1925.

On the paternal side he was descended from a long line of English Quaker ancestors, the first of whom to come to this country was Isaac Starr, who settled in Wilmington, Delaware, in 1710. Eight generations following have had sons bearing his name; the last two, Isaac Starr, a nephew of Louis, and his son, Dr. Isaac Starr, Jr., of Chestnut Hill, now a Fellow of the College, are living.

The descendants of Isaac Starr, of Wilmington, continued in the original faith, intermarrying with members of the Society of Friends, until Isaac Starr, the father of Dr. Louis Starr, married, in 1830, Lydia, the daughter of Pierre Ducoing, a native of Bordeaux, France. The Ducoing family had emigrated to San Domingo early in the Nineteenth Century, and lived in comfort on their plantation there until, in a sudden uprising of their slaves, the father lost his life. The mother with her young daughter fortunately escaped, and finally reached a new home in Philadelphia. It was here that Dr. Starr's father and mother met.

It has been remarked that this intermingling of French blood with that of the old English Quaker stock produced the happy combination of Old World charm of manner and facial characteristics that distinguished Louis Starr and his brothers.

Isaac Starr, his father, was a banker. He lived at 1417 Spruce Street, and for many years was a director of the Girard Trust Company.

Of Dr. Starr's boyhood I can give little account, for he was the last survivor of his immediate family; but a little later we find him

* Read May 10, 1926.

a student at Haverford College, where he was prominent in athletics and became the captain of the cricket eleven, an honor of some distinction in a college which has always fostered and excelled in this good old English pastime. He received the degree of Bachelor of Arts in 1868, and in passing it may be noted that his Alma Mater later recognized his professional eminence by conferring on him the honorary degree of LL.D. in 1910. Having no taste for business life, Louis Starr chose the profession in which he later made so distinguished a success, doubtless urged by some subconscious appreciation of his latent talent for the healing art. Graduated from the Medical School of the University of Pennsylvania in the class of 1871, a classmate of Arthur V. Meigs and J. William White, Dr. Starr was appointed a resident physician in the Episcopal Hospital, where he later became one of its visiting physicians.

His private practice began as a general practitioner, and he soon gained a clientèle among the prominent families of Philadelphia, many of whom continued to be his patients until he gave up active work. His love for children, however, soon turned his attention to a more intensive study of their diseases, for which facilities were offered in the Children's Hospital, then located on Twenty-second Street near Walnut. He became a chief in its dispensary, and soon was advanced to the position of visiting physician.

In 1882, then well established in his profession and already recognized as a skilful pediatrist, he married Miss Mary Parrish, a daughter of William Dillwyn Parrish, who was son of Dr. Joseph Parrish, a onetime vice-president of this College and a physician justly celebrated in his day for his self-sacrificing devotion to the people of Philadelphia during the serious epidemic of Asiatic cholera in 1832. The Parrish family was descended from Captain Edward Parrish, born in Yorkshire, England, about 1600, who had settled in Anne Arundel County, Maryland. From this union of two old Quaker families three children were born, Louis, Jr., Dillwyn Parrish, and Elizabeth Parrish Starr, of whom only the daughter has survived him.

Dr. Starr's first literary work was in assisting the late Dr. William Pepper in editing the voluminous *System of Medicine* which appeared in 1885 and 1886, to which he contributed several articles. Otherwise his writings have been confined to the subject in which he was recognized as a distinguished authority, and were published after his election in 1884 to the chair of clinical professor of diseases of

children in the University of Pennsylvania, a position he held until 1890.

In 1886 appeared his *Diseases of the Digestive Organs in Infancy and Childhood*, which reached three editions; the last, largely rewritten, was published in 1901.

In 1888 came his widely known *Hygiene of the Nursery*, the first work of its kind to achieve popular recognition. It was a model of concise, practical advice to mothers, and has served as the groundwork of all similar publications of its class. The eighth edition appeared in 1913.

In 1889 was issued under his editorship the second American edition of J. F. Goodhart's *Guide to the Diseases of Children*.

In 1894 was published *The American Text-book of Diseases of Children by American Teachers*, under Dr. Starr's editorship, with a second revised edition in 1898.

And finally, in 1915, written after his retirement, his last book, *The Adolescent Period: Its Treatment and Management*.

I first met Dr. Starr about 1890, when I became his assistant in editing the *American Text-book*. The association thus begun was continued in his private practice during the balance of his professional life in Philadelphia. It is, therefore, with a profound sense of admiration and affection for him that I welcome the privilege of presenting tonight this tribute to his memory.

Louis Starr was a born physician—a keen observer, an inspired diagnostician, a resourceful therapeutist. He was one of the first physicians in Philadelphia to recognize the value of diphtheria antitoxin and to employ it in his practice. His original methods in the feeding of infants, though based on his own purely empirical observation, before the era of percentage theories and calory calculations, proved their accuracy by their gratifying results in practice. Hundreds of Philadelphians now grown to adult maturity, it truthfully can be said, owe their lives to his skilful ministrations in their early infancy. His grasp of the vital features of a case was sure and convincing; his decisions quick and unerring; his attention to detail thorough and comprehensive. His gentle, sympathetic manner won instant response; his patients adored him.

His capacity for work was enormous. While at the height of his professional activity he often saw fifty to sixty or more patients a day, no case was neglected; no necessary detail was slurred over or forgotten.

Many of the older Fellows of the College will remember his distinguished personal appearance, happily caught by the artist De Camp in his portrait which hangs on the wall of the medical building of the University, and by Grafly in the bronze bust that adorns the niche at the entrance to this Hall. They will recall his immaculate, fashionable habit of dress; his cordial, old-time courtesy of manner; his sterling honesty of thought and speech, and his loyal devotion to his friends.

As a consultant he was ideal, holding up the hands of the physician who had called upon him for help and leaving slip no incautious suggestion of criticism that could unsettle the confidence of the family in their own medical attendant.

A remark he once made to me, when, after a long day's work, I had asked him if he were too tired to see a serious case with me—"I am never too tired to see a sick child"—expressed in a striking way his strong sense of responsibility as a physician and his gentle sympathy for all suffering humanity.

From 1890 until his enforced retirement in 1911 he worked at high pressure during the fall, winter and spring months, seeking a much-needed rest and recreation over the summer, which was usually passed at his cottage at Islesboro, Maine. But the demands upon his vitality began to tell. During a vacation spent in England in the summer of 1911 he suffered an acute dilatation of the heart, from which he gradually recovered to face the verdict of his physician, Dr. Percival White, of London, that he must give up any thought of future active professional work. Unconvinced, he came home determined to stand by the final opinion of his friend, Dr. John H. Musser. To his sorrow and, as it seemed, humiliation, Dr. Musser could only confirm the advice of the English consultant. And thus, at a word, ended the professional life of Louis Starr, and Philadelphia mourned him as if he had actually passed away.

After a few months occupied in putting his house in order, he sailed for England, where Mrs. Starr had relatives, and passed his time between London and Devonshire. He several times revisited Philadelphia for brief periods and was spending the summer of 1914 with his family at his country place in Islesboro, when, on the 4th of August, he heard the news of the outbreak of the war. His younger son, Dillwyn, was fired with enthusiasm to get into service, and soon found an opportunity to get to England under the Red Cross. In London, he joined an American volunteer motor ambu-

lance corps, and later enlisted in the English armored car division of the Royal Flying Corps, and saw service in France. Dr. and Mrs. Starr soon followed their son abroad and remained in England during the entire period of the war.

In 1915, Dr. Starr, then living in London, became interested in relief work among the wounded soldiers, especially in those Americans who had enlisted in the English service before their own country entered the war. He was constantly called in consultation by the English doctors and gave all that his strength would permit.

On the 15th of September, 1916, his son, who had passed safely through the unfortunate campaign in Gallipoli and had become a lieutenant in the Coldstream Guards, was instantly killed in a gallant charge at the head of his men in the battle of the Somme. In memory of this son, Dr. Starr wrote and published for private circulation in the following year, *The War Story of Dillwyn Parrish Starr* a noble memorial. This, I believe, was the last literary effort that Dr. Starr ever penned.

In the many sad weeks of readjustment following this overwhelming bereavement, Dr. Starr began, at first idly, almost unconsciously, to develop an unsuspected talent that was destined to bring him an absorbing interest for the remaining years of his life. Standing listlessly at his window in London one day, he began to sketch the outlines of distant roofs and chimneys. He was intrigued by the resulting picture, and made further trials. From this chance beginning he continued to practice with growing enthusiasm in sketching from Nature. He had never had any instruction in drawing: his methods were his own selftaught experiments with pen and pencil. But the soul of the artist must have waked within him and expanded with every fresh effort. Later, he sought a more facile medium of expression in the etcher's needle, and he soon acquired a delicacy of touch that gave his work the distinction of a master. In 1920, the Royal College of Physicians in London, which had made him a Fellow in recognition of his war service, held an exhibition of the artistic works of physicians, and Dr. Starr's etchings were given a conspicuous place on the line. Later, examples of his work were shown at an exhibition of the Academy of the Fine Arts in Philadelphia, where they attracted favorable attention and brought flattering offers from New York art dealers. Needless to say they were resolutely refused.

In 1921 his elder son died in London, and this second bereavement

decided him to leave England and go to Dinard, on the coast of Brittany, where he spent the last years of his life. Dinard proved an inspiring milieu for his work, for which he whimsically lamented "the days were all too short." His studio was the rendezvous of artists and American visitors who came to Dinard. Many of his old Philadelphia friends journeyed from Paris especially to see him. His home was the center of attraction for the young people of the neighborhood. They all loved him, and found in him a sympathetic advisor, a delightful comrade. These interests and his art kept him active and content. Up to within a week of his death he walked as firmly and erect as he had for years past. He was full of enjoyment of his life, and appeared to have no premonition of his approaching end. He was critically ill only a few days. He died peacefully with his hand in that of his beloved wife and constant companion, in the land of his mother's birth and of his son's heroic death. Thus passed a noble gentleman, a wise physician, a prince among men.

[As a permanent memorial of the work of his hand, I am requested by Mrs. Starr to present to the College this print of an exquisite etching, the last and most finished of a series of truly remarkable contributions to contemporary art.]

MEMOIR OF GEORGE ARTHUR PIERSOL, M.D.*

By GEORGE FETTEROLF, M.D.

In this brief contribution there will be no biographic data, nor will there be an enumeration of scientific and academic achievements. Rather, an affectionate but inadequate attempt will be made to draw a few outline sketches to suggest Piersol, the man, to those who did not know him and to help recall him to those who did.

All his life a teacher, it is in that capacity we think of him first. To the students he was "Daddy" — "Daddy Piersol." That title of "Daddy" was all affection. He knew that he was so called and he occasionally would make humorous reference to it. He accepted it in the spirit in which it was bestowed, and he gave the impression of liking it.

With the students he invariably was approachable and sympathetic. He appreciated their difficulties and was eager to help them, in personal as well as in academic matters. Until the years became burdensome it was his custom to spend Wednesday afternoons in the dissecting room. He would bring with him his after-luncheon cigar and stroll from table to table, chatting with this man or that, noting points in the dissections, answering questions, calling attention to interesting details, consulting with his demonstrators, and being in general a stimulus to teachers and students alike. Wednesdays were red-letter days in that dissecting room!

One of his great trials was the reading and marking of examination papers, a Herculean task. Descriptive anatomy being so largely a matter of memory, he would frame his questions in such a way that they were tests of ability to think as well as tests of memory. Failure to show the former registered more unfavorably with him than did evidence of lack of the latter. An instance of this, as well of his occasional use of colloquialisms, was the following:

* Read May 10, 1926.

I was working at a table in his room one day, while he was busy at his desk reading examination papers. I noticed that he was getting a little restless and that an occasional sigh would escape him. Finally he leaned back in his chair, turned around and remarked in a tone all of sorrow and none of anger, "They don't use their beans."

He was a striking exemplar of the advantages of system and order. Literally, he had a place for everything and everything was in its place. His charts, his lantern slides, all of his paraphernalia, his cigar cutter, his pipe cleaner, all were carefully placed. It therefore was never any trouble for him to get out the materials for his lectures. He spent many years in the collection and preparation of drawings, specimens and lantern slides wherewith to illustrate his talks, and, in addition, few lectures passed without his drawing on the blackboard one or more of what he always called "rude diagrams." Slowly elaborated, to permit of ready copying by the students, with great beauty of line, with pleasing choice of contrasting colors, and when finished telling a complete and simply told tale, these diagrams were models of instructive presentation. Those who attended his lectures will recall his dignified entrance and his usual opening remark: "Gentlemen, you will remember that at our last meeting we were discussing"——whatever the subject was. Then came the lecture, always delivered without notes, and with a clearness, a philosophic basis, a vocabulary and an unfailing logical sequence that were impressive and delightful to the last degree.

The College recently had the privilege of listening to a wondrous tribute, an outstanding and memory-haunting contribution to medical biographic literature—de Schweinitz's loving memoir of Leidy. Piersol yielded neither to de Schweinitz nor to any one else in his admiration of and love for Leidy.

His introductory lecture always told the story of the progress and development of anatomy. It was a scholarly tracing of anatomic science through the centuries, and those of us who heard it many times found in it each year new food for thought. A bust of Leidy adorned Piersol's lecture room. It was situated high on the wall, back of the lecturer, above the blackboard and facing the students. The climax of this first lecture always came when Piersol ended his enumeration of the nineteenth century outstanding contributors to anatomic science by half facing around, raising his arm, pointing to the bust and saying "and our own great Leidy."

Another example of this reverence, and also of his own modesty, occurred at the time of the formation by a group of medical students of the Piersol Anatomical Society. His instant reaction when the plan was presented to him was that he would personally do all in his power to aid such a society, but would suggest that the organization should call itself the "Leidy Society." This proposition was not acceptable to the students and the Piersol Anatomical Society is now one of the flourishing undergraduate organizations in the University Medical School.

He had many memorabilia of Leidy, and at one of the annual evenings at his home, truly *noctes ambrosianæ*, which he was accustomed to give to the Piersol Society, he spread these treasures before us, and leaning back in his Morris chair, cigar in hand, he talked Leidy. None of those present will forget that evening, and the thought must have come to some of those present, I know it did to one, that those qualities of Leidy which Piersol mentioned as evoking the love of those near him were truly and strikingly exemplified in the one who was speaking. It irresistibly recalled to memory Hawthorne's story of the *Great Stone Face*.

Master of anatomy, active in science, skilled in pedagogy, wise in counsel, comprehending in friendship, gentle to errors, supporting in trials, he is of the fine ones and great who have gone beyond. Those whom we love we love for their qualities of heart. Those whom we admire we admire for their qualities of mind. This is why George Arthur Piersol commanded not only the admiration but the love of those fortunate ones who received the priceless gifts of his trust and friendship.

MEMOIR OF I. MINIS HAYS, M.D.

By WILLIAM J. TAYLOR, M.D.

Isaac Minis Hays was born in Philadelphia on July 26, 1847, the son of Dr. Isaac Hays, of Philadelphia, and his wife, Sarah Minis, of Savannah, Georgia.

He received degrees from the University of Pennsylvania, of Bachelor of Arts in 1866, Doctor of Medicine in 1868 and Master of Arts in 1869. He was associated with his father who had been for many years editor of the *American Journal of the Medical Sciences*, and succeeded him as editor of this journal in 1878 and continued as its editor until 1890. He was also editor of the *Medical News* from 1878 to 1889. He wrote a number of articles upon ophthalmology and edited the American edition of J. Soelberg Wells' *Treatise on the Diseases of the Eye*, which appeared in 1873.

He was elected a Fellow of this College in January, 1872. He served as Secretary-General of the International Medical Congress which met in Philadelphia at the Centennial Exposition in 1876. He was elected a member of the Library Committee of this College and served for many years, until December, 1893, as its chairman. Up to that time he devoted much of his energies toward building up and systematizing the library of the college, but shortly after, in 1894, he became deeply interested in the American Philosophical Society, of which he had been a member since 1866, and became its secretary in January of 1897, as well as librarian, serving in this dual capacity for twenty-five years, and resigning in 1922. He was largely responsible in bringing to life the American Philosophical Society, which was almost moribund, and by his energy, systematic control and general efficiency helped materially in bringing it to its present state of activity. He was a member of the Association of American Physicians and various other societies. The College is greatly indebted to him for his interest and advice as a member of the Library Committee. He died on June 5, 1925.

As editor of the *American Journal of the Medical Sciences*, he had a wide acquaintance among medical men and showed rare judgment in selecting papers for publication in the journal.

He was tall, very upright, and dignified in appearance, courteous in his manner and an agreeable conversationalist, but very positive in holding to his own opinion and insistent in carrying through his own plans, and while in the main he was usually right in his conclusions, his methods did not always tend toward harmony in committees.

His greatest service has been to the American Philosophical Society, where, as librarian, he collected together and accurately catalogued the great mass of Benjamin Franklin's papers and manuscripts. In 1904 he published the *Chronology of Benjamin Franklin*.

ANNUAL ADDRESS OF THE PRESIDENT FOR THE COLLEGE YEAR OF 1925*

By HOBART AMORY HARE, M.D.

In accordance with the rules of the College, I am presenting herewith the address of the President. It was evidently the intention of those who were active in the early days of this venerable institution that by this means the Fellows would be given information in regard to its career during the past year, in respect to its affairs, financial and otherwise, and when opportunity offered to bring forward suggestions that might be considered as advantageous in maintaining its efficiency and usefulness not only to ourselves but to the general profession as well. Nor is it to be forgotten that the intent of the Founders was that at proper times the College should extend its activities in such a way as to prove directly useful to the community at large.

Owing to the revision of the *Ordinances and By-Laws*, much of the so-called business of the College heretofore transacted in general meetings has been transacted by the Council, and this new plan so far has seemed as advantageous as hoped for, there being at present only two meetings of the College devoted to business, namely, one in January and another in May, it being the rule that at the regular December meeting no business save nominations for officers for the ensuing year shall be transacted. For this reason there are only two meetings at which Fellows may be elected, and as the name of a candidate must be posted for six months, considerable time may elapse before his name is finally acted upon.

I desire to call the attention of the Fellows to a matter which I believe must be given serious consideration. I refer to the so-called scientific business of the College. Many years ago the number of medical societies was relatively few, whereas at present they are so

* Read January 11, 1926.

numerous that practically every night for many weeks is filled, not alone by the program of one organization, but sometimes several bodies meet on the same night. The organization of the various so-called Sections of the College has added to this aggregation, with the result that in many instances papers are produced for the College by request and sometimes only after persistent supplication and prayers. Several decades ago there was practically no competition for papers, and at that time communications usually were offered because the writer thereof had something worthwhile to present. A paper produced under the urging of a committee all too frequently lacks this quality and, however educative it may be to a certain number of medical men, it is not the type that is suitable for presentation to this body, the place of which in the medical world makes such papers inappropriate. I am glad to say that the Fellows as a rule recognize that a presentation which might be suitable to a branch of the County Medical Society is unsuitable to Mitchell Hall.

This somewhat long preamble brings me to what a former Fellow was wont to call the "point of impact," namely, as to the wisdom of making the Committee on Scientific Business fill the monthly program with three or four papers when the officers of the Sections are constantly after the same sources of supply. Medicine has become so specialized that it is difficult to prepare a program which will prove of interest to a considerable number of the Fellows, and the specialist prefers to present what he has of value to his coworkers rather than to a mixed audience. Although it was one of the early Sections permitted to organize under our rules, I have always thought that the forming of the Section on Medicine was an error in that it acts as a competitor to its mother, the College. If it chances to have a very energetic chairman and clerk it may, and has in the past, presented better programs than those of the College itself, yet the subjects suitable to its sessions are the ones most likely to bring a large number of hearers to a regular meeting of the College.

Last of all, the very superfluity of papers and meetings results in such an *embarras de richesse* that, to change the metaphor, the busy man is satiated and then hopeless of attending so many meetings. To put matters in another way, some of the best-equipped men get weary of making contributions or attending meetings and become sterile.

I cannot avoid the feeling that the papers which are read before this body should be voluntary, not written to order, and that it places the College in an undignified position to go about begging for communications. Within the last year, on one occasion no less than fifty-four Fellows were approached with the request that a paper be prepared, with not a single favorable response, and on another occasion thirty-four Fellows were appealed to with equally barren results. If the Fellows all knew that when a paper is on our program it means that the author feels he has something noteworthy to communicate, they would be more likely to believe it is worth while to attend.

It need not be pointed out that the College of Physicians is different from the ordinary medical society. In such bodies the papers presented are the whole thing, whereas in our case the programs, be they never so good, form a very small part of the function of the College. Its place is that of a nucleus representative of the dignity of the profession, its library is a source of scholarship and learning, its museum and its various collections are, all of them, the reasons for its existence.

Without undue pride I presume it may be said that this College is to America what the Royal College of Physicians is to London. That worthy and ancient institution does not have meetings such as ours, but on occasion meets for necessary business and has its lectures, such as the Goulstonian, Lettsomian and Harveian. This equally worthy College has lectureships also and they are nearly as numerous. The Weir Mitchell Lecture, the Mütter Lecture, the Hatfield Lecture and the Mary Scott Newbold Lecture are now all of them superimposed as extra occasions upon the regular programs. Would it not be well that these be given at the regular meetings whenever possible? Furthermore, the Committee on Public Health and Preventive Medicine might readily arrange for some distinguished sanitarian to give what might be called the Anders Lecture. If so desired, it might be required of the winner of the Alvarenga Prize that he read his essay in condensed form upon another meeting night. This would have the advantage of letting the Fellows know the contents of the essay, a condition not existent today, and this plan would provide perhaps the chief attraction on at least five occasions, without in any way preventing Fellows from presenting papers when they have anything in mind that they feel is worthy of these surroundings.

Unless there is some objection to this plan, I shall ask the chairmen of the various lectureship committees to confer with the Committee on Scientific Business with these objects in view, but I would like the Fellows now present to give their views.

When the present presiding officer entered upon his duties he took the liberty of making certain introductory remarks, and I again take the liberty of repeating what was said then as to the duties of Fellows in relation to making propositions for Fellowship. To quote what was said before, in many instances it has seemed as if proposers for Fellowship sometimes sign the official blanks because they are asked to, and often for no other reason than this, or because of the fact that they know of nothing *against* the candidate. Often this is done because as the Council is to act in the matter. To use the vulgar expression of the day, they "pass the buck" to that body, and when the Council fails to recommend the person his proposers feel annoyed because their signatures seemingly have not carried enough weight to ensure a recommendation for Fellowship. I make bold to assert that the primary responsibility rests upon the signers, that is, the Fellows themselves, and that until those who are asked to sign regard this act not as a favor rendered to a friend, but as the duty of a trustee of a great institution, not only made up of buildings and grounds and a great library, but also designed to uphold certain high ideals and standards in our profession, the institution will suffer. The question to be asked himself by each signer of a proposition is not, "Is this a good thing for the candidate?" but rather, "Will the election of this man be a benefit to the College?" The question is not, "Is there any reason why this man should not be elected?" but rather, "Is there one, or are there many, reasons why he should be elected?"

Large as the Council is in numbers, it is not possible for it to know of the career of each person proposed. If his accomplishments make him worthy, and the Fellows proposing him believe him qualified, they should take enough interest, not in the candidate but in the College, to communicate with several councillors and give them facts. When this is done much humiliation for proposer and candidate may be avoided, because the man will be practically sure of recommendation unless evidence is presented, unknown to to the proposer, which acts as a bar.

The question has been asked what constitutes qualification for Fellowship? This is now answered by the College Council, which I quote as follows:

1. That the candidate for Fellowship should not be considered qualified because of his promising future, but rather because of his accomplishments.

2. That in addition to qualifications as to character and behavior, stated in the By-Laws (Chapter I, Section 5), the candidate should have made some contribution of recognized value to medical literature, or that he be actively engaged in medical teaching, or that he hold some hospital position of importance, or that he should have shown outstanding professional ability as a practitioner.

3. That a Fellow who signs a proposition for Fellowship shall feel it his duty to inform members of the Council of his reasons for making the proposition in addition to the formal letter which must be sent before the Council takes action.

In previous years it has been the custom at the Annual Meeting for the various officers, such as the Treasurer, Secretary and the Chairman of various committees, to read their annual reports. Under the new rules these reports are to be made to the Council, and they were so made at its last meeting. It is manifest, however, that the College at any time during the Annual Meeting may call for the reading of any or all of these statements should it so desire, and these reports are now on the Secretary's desk to be called for if any Fellow makes the request.

Among the other changes which the College deemed it wise to make as to the programs of its meetings was to direct that memoirs were to be read only at the annual meeting in January and at the May business meeting. It is to be seen at once that several lengthy memoirs might consume a whole evening. At the present time, under the rules, it is the custom of the Censors, on the death of a Fellow, to recommend that his passing be noticed by the reading of a memoir by a Fellow of the College, or that his demise shall be mentioned in the President's annual address.

In accordance with the recommendations of the Censors, various Fellows have been asked to prepare memoirs, until at present twelve such memoirs are listed, but in each case the Fellow who has accepted the task of preparing the memoir has as yet failed to do so. Incidentally I may add that in one instance the memoir has been due since 1918 and another since 1920. The result of this is that if the President includes in his address mention of those who have been not so prominent as to deserve a memoir, their passing is officially brought before the College, whereas the passing of a more prominent

Fellow is entirely ignored. For this reason, I announce all the deaths during the year 1925 of the following Fellows, without separating them into groups:

Dr. Charles B. Penrose, elected April 3, 1889. Died February 27, 1925. Memoir of whom was read on October 7, 1925, by Dr. Edward Martin.

Dr. John B. Turner, elected June 5, 1901. Died March 3, 1925.

Dr. I. Minis Hays, elected January 3, 1872. Died June 5, 1925. Dr. Hays was for many years an Active Fellow of this College and Editor of the *American Journal of the Medical Sciences* and the *Medical News*. A memoir will be prepared by Dr. William J. Taylor. Dr. Hays was on the Library Committee of the College from 1873 to 1893, and chairman of that committee from 1887 to 1893. In 1882 he was elected to the Committee on Publication. He was appointed by Dr. J. M. DaCosta, during his second period as President, a member and chairman of the Committee on Finance, serving as such until 1900, during the administration of Dr. Ashhurst and the first two years of Dr. Keen's presidency. He also served the College as a delegate to the convention on the revision of the U. S. Pharmacopœia in 1880.

Dr. Rufus B. Scarlett, elected October 5, 1919. A young man whose labors in the practice of rhinology and laryngology were full of much promise. Died July 11, 1925.

Dr. Louis Starr, elected April 7, 1875. Physician to the Episcopal Hospital; later clinical professor of diseases of children in the University of Pennsylvania. Died September 12, 1925. A memoir will be read by Dr. Thompson S. Westcott.

Dr. John C. Hirst, elected November 4, 1903. An active practitioner in obstetrics and gynecology, and the author of a well-known book on obstetrics. Died October 4, 1925.

Dr. Louis Jurist, elected February 3, 1886. For nearly forty years a Fellow; an active laryngological practitioner, although a rare attendant at the College meetings. Died October 9, 1925.

Dr. John H. W. Rhein, elected February 3, 1897. He was actively engaged as a general practitioner and neurologist, receiving his early training in the latter department of medicine under the late Dr. S. Weir Mitchell. Died October 14, 1925.

Dr. John Guitéras, elected in December, 1889, and a Corresponding Fellow October 4, 1916. A distinguished graduate of the University of Pennsylvania and professor of pathology in that

institution; famous also as an authority in tropical diseases and as a sanitarian not only in this country but in his native land (Cuba), to which he returned in the later years of his life. Died October 28, 1925.

Dr. Richard H. Harte, elected May 6, 1885. An able surgeon to numerous hospitals; active for a number of years in the teaching staff of the University of Pennsylvania; distinguished for his career as a medical officer during the World War, and a generous donor not only during his life but by his will to the College of Physicians, of which he was President from 1916 to 1919. Died November 14, 1925. A memoir of Dr. Harte is about to be prepared.

Dr. Edward W. Watson, elected March 3, 1886. An active practitioner for many years in the field of laryngology; a contributor to medical literature in his earlier years to a considerable extent. He also made contributions to nonmedical literature. Died November 20, 1925.

Dr. Henry R. Wharton, elected October 1, 1884. For many years an active teacher of surgery in the University of Pennsylvania; surgeon by appointment to many important hospitals, notably the University, the Presbyterian and the Children's Hospital; author of an excellent book upon *Minor Surgery and Bandaging* which has gone through many editions. Died December 3, 1925. A memoir is to be prepared.

Dr. David Gregg Metheny, elected a Fellow in 1923; at the time of his death professor of anatomy in Temple University, who died December 15, 1924.

Dr. James V. Ingham, for many years deeply interested in this College, faithfully serving it for a long period of time on the committee of the Directory for Nurses, who died December 29, 1924.

It is evident that either the practice of reading memoirs had better be abrogated, or that some means of urging the Fellow who has accepted the task of reading a memoir be devised.

As already indicated, the next meeting at which memoirs can be read will be in May. If the Fellows already appointed should report that they have their memoirs ready for that meeting, it is evident that a goodly proportion of its time will be devoted to such memoirs, with little time for the transaction of College business, the more so if Fate during the next few months causes the Board of Censors to recommend other memorial contributions.

I mention these facts not to chide those who have failed to carry

out an accepted task, but to call the attention of the College to the question as to whether they wish the Board of Censors to make such recommendations in the future.

As a substitute for the actual reading of a memoir, possibly it will be the wish of the College to rule that, except under unusual circumstances, it will be adequate to publish the memoir only in the *Transactions*. I would ask the Fellows to express their views at this time as to this matter. No fixed rule need be laid down and on rare occasions it may be well to have a memoir read when it deals with the career of some especially noteworthy Fellow who has passed away during the preceding twelve months.

During the past year the following noteworthy gifts have been made to the College: $394.02 from the estate of Charles A. Oliver (Library Fund), this amount due the College at death of his widow. $489.73 from the estate of Elizabeth R. Moulton for Charles T. Hunter Library Fund, on account of $5000 left in a codicil to the College, the same to be paid out of trusts to annuitants as each one dies ($500 less tax). $1000 from Dr. E. E. Montgomery, his third $1000 toward a $5000 Library Fund; $5000 from the estate of J. William White for books on surgery (estate now distributed on death of his widow); $600 from Mr. Clement B. Newbold, annual contribution for the Mary Scott Newbold Lecture.

ANNUAL ADDRESS OF THE PRESIDENT FOR THE COLLEGE YEAR OF 1926*

By HOBART AMORY HARE, M.D.

Under the ordinances of the College it is the duty of the President at this meeting to make an address in which he presents a summary of events during the past twelve months, makes recommendations as to changes which may be considered by the Fellows, and informs the College of certain facts which, heretofore, have been presented by other officers or the Chairman of the various Committees. Under the new rules the various committees report to the Council.

The finances of the College are in excellent shape, although at times it is necessary to be most careful and economical in the expenditure of funds. A number of sources of income are so strictly limited by the terms of the bequests that they cannot be applied except to a single purpose. I would suggest that an effort be made, when Fellows are able to interest prospective givers, to have contributions or bequests made to a maintenance fund. It is entirely possible for this to be done in such a way that the name of the donor appears as clearly as it does today when attached to one of the existing special funds. Thus a particular gift could be called the John Brown Fund for Maintenance of the College.

The College possesses a valuable property in its old building and lot at Thirteenth and Locust Streets, and when it has been sold will have added to its general fund several hundred thousand dollars. This area of the city for a time after it was vacated by the College was in relatively little demand, but I am glad to report that values in this district are rapidly increasing in value. The Finance Committee is fully alive to the fact that an excellent price should be obtained for it.

* The President's Address covering the College year 1926 was delivered before the College January 10, 1927. This volume was then being prepared for publication and the Committee of Publication considered it advisable to include the address in the present volume, as it will be of greater timely interest to the Fellows of the College, rather than to defer its publication to the next succeeding volume, a year hence.—[Editor.]

Under the new Rules and Regulations of the College, the annual reports of the Officers of the College, including reports of the various standing committees are not read, unless called for at the January meeting. For this reason I am including in my address certain facts contained in these reports which are of interest.

The Secretary states that during the year there were nine stated meetings, two business meetings, and two special meetings; that the average attendance was 40, a decrease of 10 from the previous year; twenty-eight papers have been read at the meetings of the College, fourteen of which were illustrated with lantern slides.

The Nathan Lewis Hatfield Lecture was delivered at the special meeting of February 13, by Dr. Knud Faber, of the University of Copenhagen.

No lecture under the Mary Scott Newbold Foundation was delivered in 1926.

There were three memoirs read: That of Dr. George A. Piersol by Dr. George Fetterolf; Dr. Louis Starr by Dr. Thompson S. Westcott, and Dr. Richard H. Harte by Dr. George W. Norris.

The Mütter Lecture for 1926 was delivered by Dr. Walton Martin, the professor of clinical surgery in Cornell University.

A number of rare and valuable books and photographs were presented to the Library.

A legacy of $15,000 was received from the estate of Mr. Clement B. Newbold, the income of which is to be used for lectures in memory of Mary Scott Newbold; also a legacy of $10,000 from the estate of Dr. Richard H. Harte, the income from $5000 to be used for the purchase of books and the income from the remaining $5000 for the entertainment of noted speakers asked to read papers before the College.

There was also received from Dr. E. E. Montgomery the third $1000 toward his library fund of $5000.

The Alvarenga Prize for 1926 was awarded to Dr. P. S. Pelouze and to Dr. Frederick S. Schofield.

At the meeting of January 11, an amendment of the By-Laws was carried, making the entrance fee for Fellows $30, no entrance fee being exacted of Fellows who may be reëlected, and to the effect that the annual dues shall also be $30.

The Council had nine stated meetings, and all matters referred to it by the College have been considered.

At the September meeting of the College the President appointed

a Committee on the Lister Centenary Celebration to be held April 6, 1927.

Twelve proposals for Fellowship were acted upon, 9 were reported favorably for election, 1 was withdrawn, and 2 postponed.

On December 1, 1926, the number of Fellows was 501, Associate Fellows 23, of whom 16 are American and 7 foreign, making a total of 524. Of the 501 Active Fellows, 459 reside in Philadelphia or its immediate vicinity in Pennsylvania, and the remainder are nonresidents.

The Committee on Publication reports for 1926 that during the year the *Transactions* for 1925 were published. It is a large volume of 917 pages with numerous illustrations, and contains a very complete record of the activities of the College.

A point of very considerable importance for consideration on the part of the Fellows of the College is the question as to whether it is wise for the College to continue publishing a large volume of *Transactions* which each year is becoming larger and more expensive, chiefly because the *Transactions* of the Sections are included. The cost of printing the *Transactions* amounts to $4207.90. It has been wisely pointed out that very few of the Fellows read the *Transactions* and that the papers appearing in the *Transactions* are buried, or, if not buried, only escape because they are published elsewhere in some current medical publication in order that the authors may bring their views before the general medical profession. It is thought that perhaps $2000 a year might be saved by publishing merely the list of Fellows with the By-Laws and programs of the various meetings of the College and of its Sections, and, if this were done, a reference after each title of a paper could state the journal in which the author intended to publish his communication.

The second proposition which has been advanced is that the lectures presented before the College be immediately printed in the form of what is commonly called a "reprint," which might be bound with a dignified cover, have the seal of the College impressed upon it, and such a heading, for example, as "Transactions of the College of Physicians, fasciculus 32, Mary Scott Newbold Lecture," then giving the title and the author. A sufficient number of these could be printed so that at the end of the year one of each of the variously endowed lectures could be put in what might be called the outline *Transactions* of the College, or they could be mailed immediately upon being printed to the Fellows, who could preserve them or not as they chose. This would have the additional advantage that the

lecturer could be provided, with or without expense to him, with a reprint of what he had presented, and if any considerable number was desired he would bear the expense. It is manifest that such a procedure would not only save the College several thousand dollars, but would place in the hands of the Fellows who read papers and visitors who give lectures a copy of their paper or lecture so that it might be distributed while the topic remained a live subject. Under such an arrangement, also, the President's address might be included, not only in the bound *Transactions* but form a separate fasciculus, to be immediately distributed. This point will be discussed later in this address.

If these suggestions are carried out, the titles of the various papers with the names of the authors and the references to the journals in which the articles are published, then those who receive a bound copy of the *Transactions* for the year would readily discern what the College had been doing during the past twelve months, and if any reader was specially interested he could readily turn to the journal containing the full article if he wished details.

In previous years it has been the custom at the Annual Meeting for the various officers, such as the Treasurer, Secretary and the Chairmen of various committees to read their annual reports. Under the new rules these reports are to be made to the Council, and they were so made at its last meeting. It is manifest, however, that the College at any time during the Annual Meeting may call for the reading of any or all of these statements should it so desire, and these reports are now on the Secretary's desk to be called for if any Fellow makes the request.

The Library Committee makes the report of a total number of volumes in the Library, including bound volumes and 21,823 unbound reports and *Transactions*, 149,739; the number of unbound theses and dissertations is 26,397, and the number of unbound pamphlets 163,786. Included in the above are 4305 volumes known as "reserves," consisting of second copies of some of the more important periodicals; also 2864 volumes more or less incomplete. The duplicates, which number 2509, are not included in the above total. The details of the report of this Committee will be published as usual and distributed to the Fellows of the College at a later date.

Suffice it to state, the College Library is the second largest medical library in the United States, the first being that of the Library of the Surgeon General's Office, and that its incunabula form one of the most remarkable collections in existence.

The report of the Committee on the Mütter Museum points out that its contents are little known to the Fellows and readers at the Library, and recommendations are made that further facilities should be afforded for the use of these exhibits at the meetings of the College or of its Sections. It is also pointed out that students and their instructors use the Museum too little, and it is recommended that a catalog of the contents of the Museum be provided, that the Curator should be paid a special honorarium for this work, and that the expense of printing the catalog be met by its sale at cost price to those interested. During the year a number of interesting gifts have been received.

The Committee and its new Curator have begun a complete renovation of the Museum.

The Committee on the Directory for Nurses reports a total registration of 986; total number of nurses registered during the year 209; total number of calls filled 8325.

The Committee on Entertainment reports that there have been no entertainments during 1926 under its supervision.

The report of the Committee on Scientific Business has already in part been covered by the report of the Secretary, giving details as to papers read and Fellows present.

It is the intention, with the approval of the Council, to place several of the annual lectures upon regular meeting nights.

The report of the Committee on Public Health and Preventive Medicine is to the effect that Dr. William H. Park, of New York, on April 7, discussed "The Control of Diphtheria and Scarlet Fever."

The report of the Committee on the Alvarenga Prize and the Nathan Lewis Hatfield Lectureship have already been referred to.

Time does not suffice to give details as to the Proceedings of the various Sections. An examination of the reports made by these Sections, which will appear later in type, will give an adequate idea of the excellent work done by the members.

During the year the following Fellows have died:

January 4, Dr. S. Lewis Ziegler.
January 24, Dr. Edward G. Rhoads.
August 18, Dr. Allen J. Smith.
September 1, Dr. William A. Hitschler.
September 26, Dr. Wilmon W. Leach.
October 4, Dr. H. McVeigh Brown.
November 12, Dr. Howard Dickes Geisler.

Under the recommendation of the Censors a memoir of Dr. Henry R. Wharton, who died December 3, 1925, will be read at this meeting by Dr. Jopson; another of Dr. Allen J. Smith, will be read at this meeting by Dr. Edward Martin, and a third, by Dr. W. Egbert Robertson, of Dr. J. B. Roberts.

A subject of very grave concern is the matter of contributions to the College Meetings. The establishment of sections devoted to *special* subjects was a wise move, but, if I may be allowed to say so, the establishment of a Section of General Medicine has resulted in depriving the College itself of a number of papers which normally belong to the proceedings in this Hall. The presentation of the papers before the College would not be limited to the Fellows because any reputable physician can hear them when so read, the more so as private business can no longer be transacted except at two business meetings per annum or in executive session. Those in charge of the Section on Medicine deserve much credit for their efforts, but their endeavors result in feeding the child at the expense of its mother. Papers that are worthy of the Section should be worthy of the College itself. The many medical organizations throughout the city divert attendance from the College meetings, but it is a noteworthy fact that when some topic of wide general interest is on the program this hall is filled to capacity. I state with regret that during the past year only four papers were volunteered for the College and only two seemed suitable. Yet I venture to say that if the Fellows who have read papers elsewhere had offered them here they would have been an *embarras de richesse*.

There would appear to be several possible procedures:

1. To abolish the Section on General Medicine. There may be opposition to this, but I believe it might be wise and proper.

2. To arrange that a certain number of the regular meetings be used for the various lectureships. This would cut down the number of general programs and leave a few at which Fellows could present papers, and if perchance so many papers were offered that time did not suffice, then one or more lectures could be given on separate nights, as is done at present. It is cause of keen regret that the Committee on Scientific Business has to ask Fellows to make contributions.

3. A third alternative would be to so change the rules that regular monthly meetings would be abrogated except when contributions are ready for presentation, only holding meetings for lectureships, and withdrawing from the rule of monthly transaction of

scientific business, the College being chiefly devoted to its Library and other functions. Personally I would be opposed to such a plan.

If so be that the President's address is to deal with things that are past, then there is no need for the discussion of things of the future, but it is evident that if such an address is of any value the present custom of presenting its contents to the Fellows should be changed by suggesting that a considerable number of Fellows feel it their duty to hear it, or at least that a copy be sent to each Fellow as soon as possible after it is delivered, or referred to the Council for action. At present, and in the past, the President's address appears in the *Transactions* a year after it is given. Thus, the address of Dr. Neilson delivered January 2, 1924, appeared in January, 1925, and his address of January 4, 1925, appeared after the lapse of many months. The address of the present holder of the office of President, read in January, 1926, has not yet, in January, 1927, come in type before the Fellows. The present address, under the present plan, will appear next December or January, when it will be, perhaps, worthily considered too old to be worth considering.

These facts are the more important because under the rules, with only two business meetings a year, no final action can be taken on many matters for six months to a year unless new amendments proposed at this meeting be acted on in May.

I, therefore, make bold to urge the Fellows who are present this evening to debate and consider whether any of the suggestions so far made have any value or are capable of alteration with advantage.

To recapitulate they are as follows:

Shall the College no longer continue the Section on Medicine?

Shall the stated meetings for Scientific Business be filled as a rule by the various so-called lectures or addresses? If so, papers that Fellows desire to read before the College can be presented only at the regular meetings which are not occupied by lectures or before or after the lecture. Shall the *Transactions* be cut down as suggested?

It may not be out of place to remind you that under the rule the Committee on Scientific Business has the power to make some of these arrangements, at least after conference with the Lectureship Committees, as a matter of courtesy. All these points, save that in regard to the Section on Medicine, are, under the rules, quite capable of determination by the Council, and much of any discussion that may follow will aid the Council without of necessity being mandatory.

SOME APPLICATIONS OF THE LABORATORY VIEWPOINT IN CLINICAL MEDICINE*

By A. D. HIRSCHFELDER, M.D.
PROFESSOR OF PHARMACOLOGY, UNIVERSITY OF MINNESOTA
(By Invitation)

The only excuse for experiments in medicine lies in the needs that arise directly in the patient, and the only excuse for an experiment in the laboratory is when it is possible to create there conditions which are much simpler than those which are met with at the bedside and which furnish us with solutions for bedside problems. The laboratory plays the rôle of the ham in the sandwich: it stands as the layer between the need met with in the patient and the final solution by application upon the patient. And so, this evening, I shall speak to you about several lines of work in which I have been interested, in which the experiments of the laboratory have thrown some light upon clinical problems. Some of these investigations have been done as research problems by undergraduate students in the Department of Pharmacology of the University of Minnesota as part of the elective work of the regular curriculum. I have seen good evidence to show that the experimental investigation of such problems has contributed materially to the education of these students, and in many cases to their subsequent success in the practice of medicine, even in the small towns.[1]

The first need at the bedside is clear-cut diagnosis, the most clinical of all the sciences. What can the laboratory add to physical diagnosis?

When we began at Baltimore to study the heart sounds by taking graphic records with the microphone and string galvanometer, Dr. Bond and I were impressed by the fact that some sounds were

* Read January 6, 1926.

[1] It must be understood, however, that the amount of such work should, as a rule, not be excessive. The total time spent in such investigations by the students referred to has been from sixty-six to one hundred and thirty hours.

heard louder by our ears and some were magnified by the microphone and sometimes the microphone picked up and recorded sounds that were very difficult to hear at all. I have found that some of this discrepancy depends upon the pressure which we exert with the stethoscope. When we press heavily with the stethoscope, we obliterate the rather low-pitched, faint sounds that are produced by the auricle, and exaggerate the high-pitched sounds of the mitral regurgitant murmur. I found that by the simple expedient of always, wherever one listens, passing through the whole range of pressure from just touching the skin with the stethoscope to pressing the stethoscope heavily in the same place, it was possible to hear a whole range of sounds which otherwise one missed. With the lightest touch you can hear the faint tick of the auricle in cases of heart-block, and with heavy pressure you bring out the high-pitched murmurs of mitral and of some aortic regurgitations. We enable our ears to do more of the things than can be done by graphic records with the microphone.

I suppose that most of you have wondered, as I have wondered, why the murmur of mitral regurgitation, which is produced by a stream of blood flowing toward the back, should be heard loudest over the apex, as though the regurgitant stream were flowing forward. A few years ago I had two of my students, Grey and De Sautelle, investigate this question by allowing water to flow through a dead pig's heart in the laboratory. They found that if they listened directly over the murmur, it was loudest over the bases of the papillary muscles; if the papillary muscles were cut away from the heart wall, the murmur at the apex greatly diminished. On the other hand, if they stitched the papillary muscle to interventricular septum, the murmur which had greatly diminished over the apex was heard loudest over the septum where the papillary muscles of the chordæ tendineæ were attached. So this simple laboratory experiment shows that the chordæ tendineæ and the papillary muscles act like violin strings to conduct the sound vibrations from the valves to the wall of the left ventricle at the apex.

We have learned an immense amount about diagnosing clinical arrhythmias with the electrocardiograph, but no one has devised a pocket edition of the electrocardiograph. So what can we do by the methods of simple physical diagnosis at the bedside? If we take what has been learned from tracings of the venous pulse and translate that into simple inspection, we can do a good deal more,

I think, than has been suggested by Mackenzie and Lewis toward accurate diagnosis—almost as much as we can in the diagnosis of valvular lesions by the stethoscope.

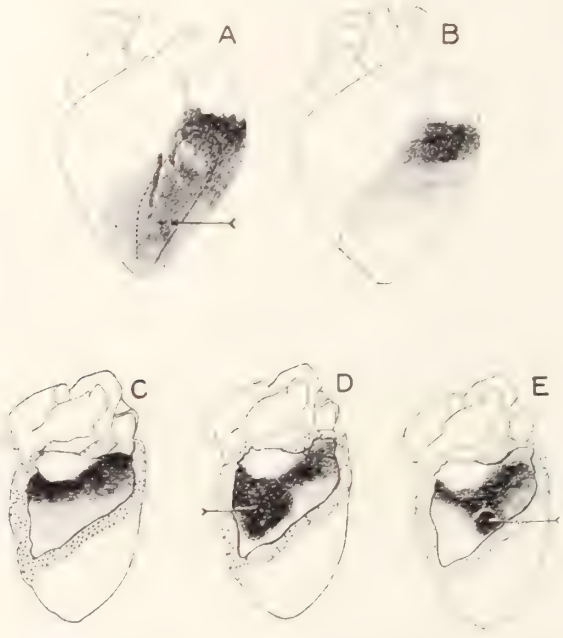

FIG. 1.—Variations in the intensity of murmur in experimental mitral regurgitation produced in the dead pig's heart, showing the violin string action of the papillary muscles and chordæ tendineæ. (Slightly modified from Grey and De Sautelle, Arch. Int. Med., 1911, viii, 734.)

The depth of shading indicates the loudness of the murmur over the area indicated. The arrow indicates the insertion of the papillary muscle or chorda tendinea. *A*, Anterior aspect of the normal pig's heart. *B*, Distribution of the murmur in the same heart after cutting away the base of the anterior papillary muscle. *C*, Distribution of the murmur over the septal surface of the right ventricular cavity after cutting away the surface of the right ventricle. *D*, Distribution of the murmur heard over this surface after suturing the anterior papillary muscle of the left ventricle to the septal wall of the left ventricle. *E*, Distribution of the murmur in the same area after suturing the chorda tendinea to the septal wall of the left ventricle.

The normal heart-beat shows itself over the jugular vein just above the clavicle in the form of two wavy indulations for each beat that we can feel in the carotid. I shall call this the double venous pulsation. We can make these waves much easier to see by just holding a visiting card within a millimeter or two of the skin just above the clavicle.

In the sinus arrhythmias, the respiratory arrhythmias, the arrhythmia which is so common that it is practically normal in children, we find the same double pulsation, but irregular in rate—usually made regular by holding the breath.

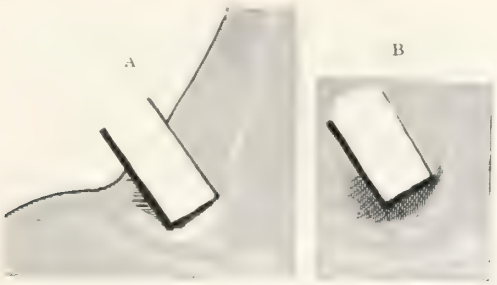

FIG. 2.—Inspection of the right jugular vein with a card held to the skin. *A*, Vein full (pulsation); *B*, Vein empty (collapse).

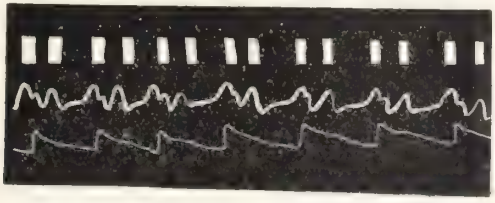

FIG. 3.—Normal (double) venous pulse. The upper line shows the pulsations, the middle line the venous pulse as recorded or observed; the lower line shows the pulsation as felt over the carotid artery.

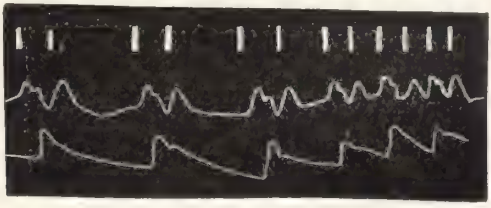

FIG. 4.—Pulsations and pulse curves in sinus arrhythmia, showing a series of slow normal (double) venous pulses alternating with a series of rapid ones.

If there are auricular extrasystoles, they usually occur as irregular occasional early beats, each showing the double venous pulse.

If there are occasional ventricular extrasystoles, you get the normal double pulse, occasionally interrupted by a single big wave

which usually sweeps up along the jugular vein to the angle of the jaw, and one can often diagnose this across the room.

If there is a heart-block, you get three or four waves over the vein for every single beat in the carotid artery. If it is a partial

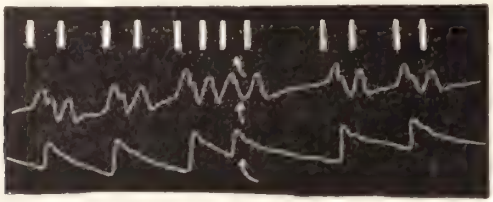

FIG. 5.—Pulsations and pulse-curves with auricular extrasystoles. The arrow points to the extrasystole which is accompanied by premature double pulsation in the vein.

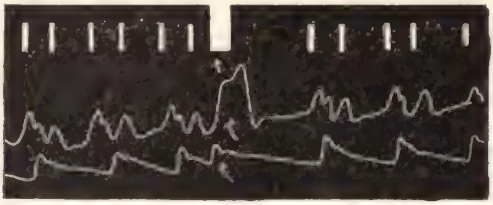

FIG. 6.—Pulsations and pulse-curves accompanying a ventricular extrasystole showing the very large single venous pulse.

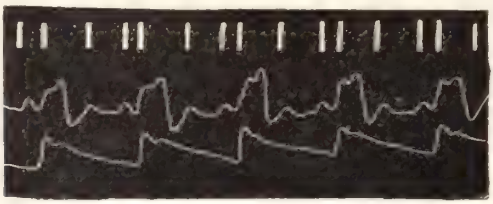

FIG. 7.—The single irregular venous pulse accompanying auricular fibrillation.

heart-block the number is always the same. If it is a complete heart-block the number of waves between successive heart beats varies from beat to beat.

If you have auricular fibrillation, you find only the same number of waves over the jugular vein as you get over the carotid, but both jugular and carotid are irregular in rate. Now as most patients with auricular fibrillation have a high pressure in their veins, you

usually have to make them sit up, or even stand, to get a good visible pulsation in the veins.

And so it is possible to make use at the bedside of a good many of the things that have been learned in the laboratory and though these simple methods do not in any way take the place of the electrocardiograph, they will help us a good deal when we cannot have the use of one.

We have all been interested in the mysterious action of quinidine in stopping the irregular contractions of the auricle in auricular fibrillation, and we all have wondered how this is brought about.

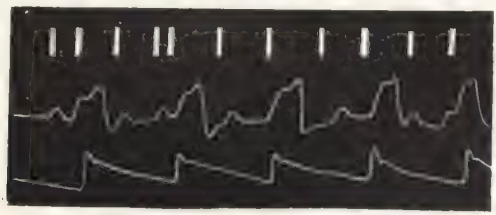

FIG. 8.—Pulsations in complete heart-block. Several pulsations for each beat in the carotid, but the number between successive carotid pulses is inconstant.

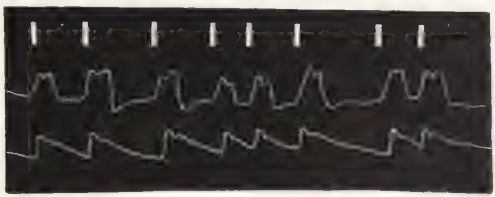

FIG. 9.—Pulsations in partial heart-block. Several pulsations for each beat in the carotid, but the number between successive carotid beats is constant.

Hoffman, Cohn and Levy, Waddell and others have shown that this drug lowers the irritability of heart muscle and seemed to somewhat lower auriculoventricular conduction. Since Garrey, Mines and Lewis have made out a good case to believe that fibrillation is due to circus movements, with new impulses arising in new parts of the heart and chasing one another about from fiber to fiber, instead of all fibers contracting at once as is the case in the normal contraction, this seemed to leave two possibilities: (1) That it decreased the tendency of abnormal foci to start or continue contracting independently of the normal focus; (2) that these foci continued

to set up abnormal contractions, but the power of the auricles to conduct them from fiber to fiber was so depressed by the drug that they no longer stimulated neighboring fibers.

Last year M. Cervenka and I investigated the action of quinidine on the terrapin's heart. We threw rapid rhythmic induction shocks into the right auricle of the terrapin's heart and we registered the contractions of both auricles. After quinidine it took a much stronger shock to cause the auricle to contract, but every time that the right auricle contracted the left auricle contracted also. In other words, even with tremendous doses of quinidine there was

FIG. 10.—Diagram showing the course of "circus movements" from fiber to fiber in the right auricle.

no markedly diminished conduction from one part of the auricle to another, but the drug greatly diminished the irritability of the auricles. This would seem to thrown preponderance to the view that quinidine acts by preventing the abnormal stimuli from arising, rather than by depressing the conduction of abnormal impulses from one fiber to another.

We have also been interested in the new respiratory stimulant, homocamfin, formerly known as hexeton, a camphor-like substance which has been prepared in Germany. It was studied in the laboratory by Gottlieb, who found that it had a camphor-like action as a circulatory and especially a respiratory stimulant, several times more active than camphor. Messrs Kasper, Kumpf, Maleride and

I studied its effects upon animals, particularly its effects on the respiratory center. We were able to confirm Gottlieb's observa-

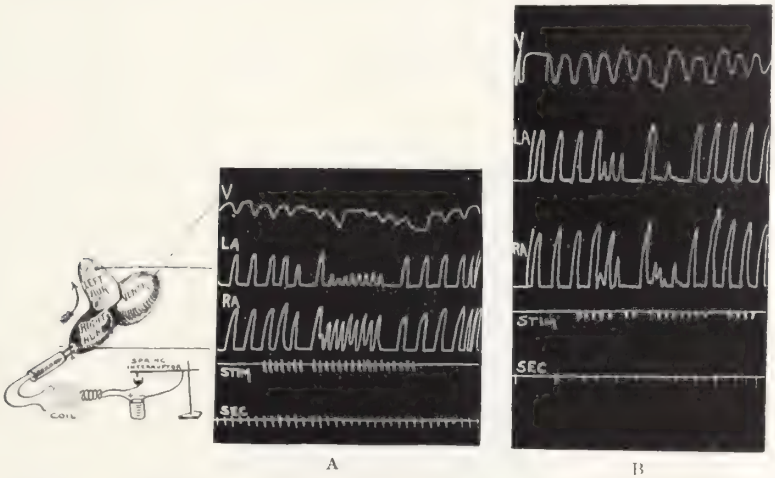

FIG. 11.—Apparatus used for stimulating the auricle of the terrapin, with curve showing the response of both auricles to the rhythmic stimuli applied to the right muscle. *A*, Before quinidin. *B*, After quinidin. (A. D. Hirschfelder and C. Cervenka, Jour. Pharmacol. and Exper. Therap., 1925, xxvi, 19.)

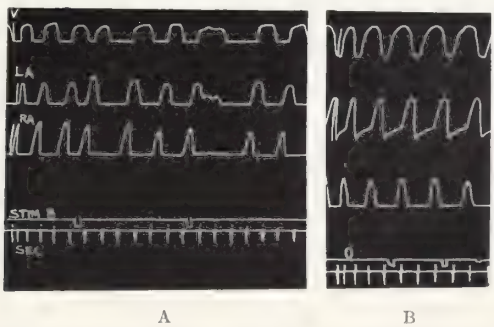

FIG. 12.—Similar record showing the reduction in irritability of the auricular auricle. *A*, Before quinidin, induction coil at 8. *B*, After quinidin, induction coil at O. (Stimulus very much greater.)

tions that it stimulates a respiratory movement which has been depressed by morphine and we found also that it does the same thing when the respiratory center has been depressed by too much

ether. So this drug ought to be particularly valuable in surgical operations in which the respiratory center shows signs of failure, as was found by Umber, Wich, Lowinger and Krehl. Dr. George Fahr has found that it is very useful in the clinic in respiratory depression in pneumonia and heart disease. Our laboratory experiments confirm the German observers that it is much more effective than camphor.

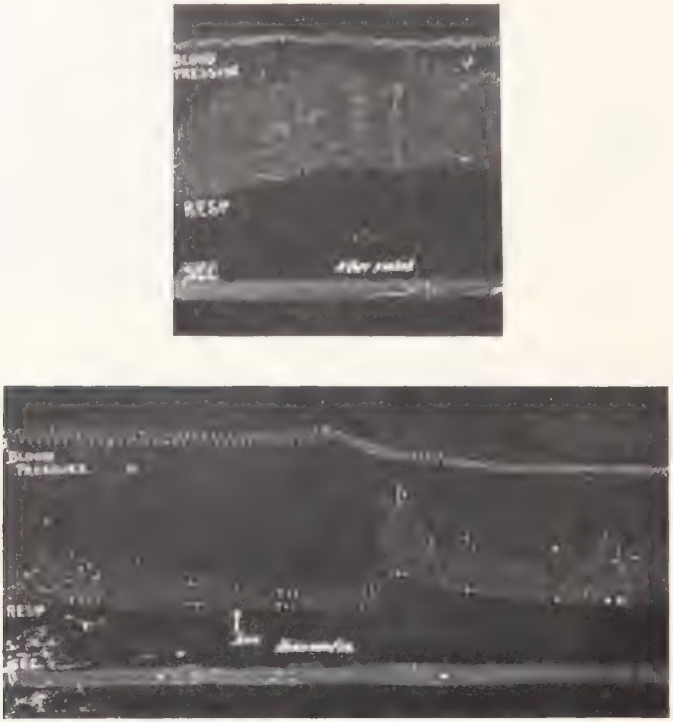

FIG. 13.—Respiration and blood-pressure curve of a dog showing cessation of respiration after morphine and resumption after homocamfin, without rise in blood-pressure.

On account of the fact that homocamfin has this effect, and that it is also a cerebral stimulant, it ought to be particularly valuable in the treatment of the barbital (veronal) and luminal poisonings, which are now becoming so common, and it certainly does increase both the rate and depth of respiration in rabbits which are anesthe-

tized with large doses of barbital; but we have as yet no data as to its antidotal actions in clinical poisoning.

Another question of importance for practical medicine, upon which we have as yet only scratched the surface, is the question of whether the ordinary clinical doses of drugs produce any actual structural alterations in the viscera, such as the liver. Our attention was called to this a few years ago by clinical observation of Dr. J. P. Schneider, who found large amounts of bile pigment in the duodenal contents of a healthy medical student who had been taking quinine in ordinary doses. Dr. Siperstein and Dr. Litman injected quinine into rabbits in amounts corresponding to 30-grain doses in human beings, and to our surprise found that these caused cloudy swelling and vacuolization of the liver cells, and somewhat larger doses caused marked degeneration. This would lead us to believe that the same thing may be true of many other drugs even in ordinary clinical doses, and that even where histological changes are not demonstrable with the microscope, many drugs are causing very marked functional changes in the interior of the cells of the liver and other glands of internal secretion, and in the permeability of their surface layers — perhaps, too, that this may affect the secretion of antibodies, either favorably or unfavorably.

I believe that the intravenous use of mercurochrome in general infections was born in Philadelphia (Piper), and as everyone is using it or has used it for septicemias or localized infections, we were interested to see whether mercurochrome would actually pass from the veins into inflammatory exudates. Accordingly, Mr. Malmgren and Mr. Creavy produced some edema in the subcutaneous tissues of the neck of rabbits by injecting the black fur dye, paraphylendiamin, which gives a peculiar collar-like edema reaching from the jaw to the clavicle. Then we injected clinical doses of mercurochrome, and about an hour later killed the animals and removed the edema fluid. It was stained a nice salmon pink with the mercurochrome and contained the drug in about the same strength as blood serum, about 1 to 12,000; but when we tested its bactericidal powers by the capillary pipette method of Drs. Kolmer, Heist and Solis-Cohen, we found that staphylococci and colon bacilli would grow beautifully in this salmon pink edema fluid. The same thing occurs in the edema of a burnt area or in the conjunctivitis produced with mustard oil. The mercurochrome penetrates into the tissues, but it does not render them antiseptic.

FIG. 14.—Similar curve showing depression of respiration by ether and stimulation by homocamfin.

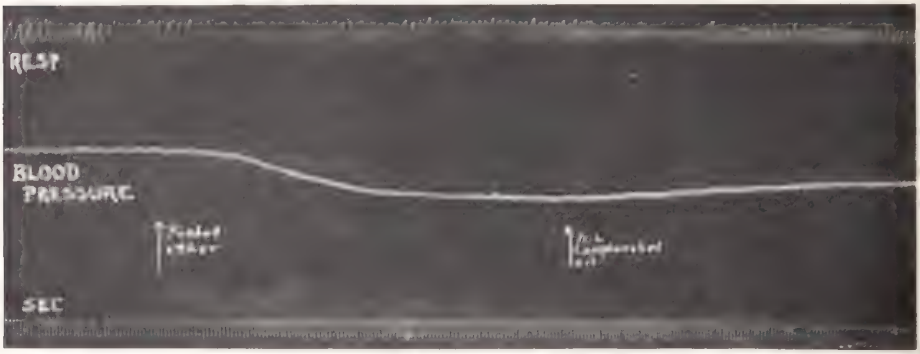

FIG. 15.—Similar tracing showing much less effect from camphor.

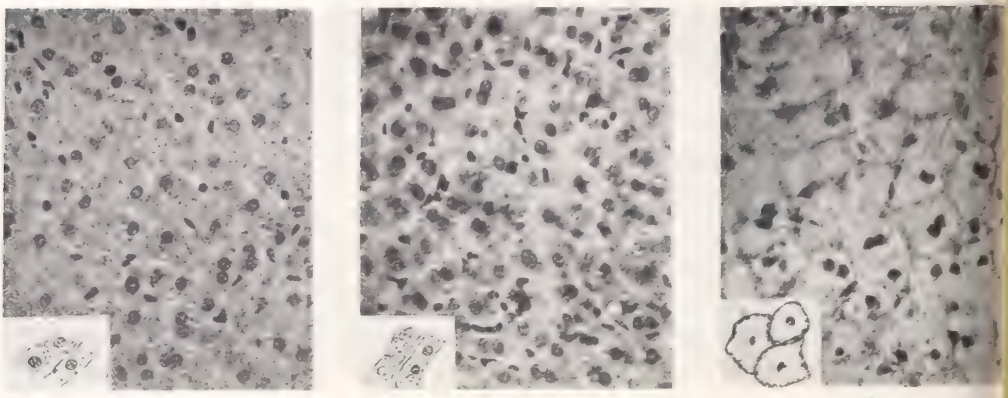

FIG. 16.—Alterations in the liver after injections of quinine. *A*, Normal rabbit's liver. *B*, Forty-eight hours after a dose of quinine sulphate equivalent to 30 gr. (2 gm.) to an adult man, showing slight degenerative changes in the liver with vacuolization and cloudy swelling of the cells. *C*, One hundred and twenty hours after large dose of quinine sulphate, equivalent to 90 grains (5.8 gm), to an adult man (82 mg. per kilo), showing very marked degeneration of the liver cells. (Siperstein and Litman, Arch. Int. Med., 1921, xxvii, 449.)

If we used acriflavine we found that the edema fluid became yellow, the dye penetrated a little more slowly and it did not render the edema fluid antiseptic.

If we used gentian violet, no dye penetrated into the edema fluid at all; it was not even present in a colorless form; at least, we were never able to regenerate it with oxidizing agents.

Recently a great deal of practical importance has attached itself to magnesium sulphate as a sedative drug. Meltzer and Auer have shown that you can put a rabbit into deep sleep immediately by a small injection of magnesium sulphate, but you can wake him up in an instant with calcium. Meltzer has shown that magnesium sulphate hypodermically can be used to suppress the convulsions of strychnine and tetanus, and in pediatrics it has been used to suppress the hyperirritability of spasmophilia. Gwathmey has given it a much wider range by showing that very small amounts when given along with morphine seem to intensify and to prolong the action of the morphine; and still further, that small amounts of magnesium sulphate, along with ether in oil, by rectum, can be used for rendering the early stages of labor practically painless. How does magnesium act? The strange thing about magnesium and calcium is that they are both antagonists of sodium in making emulsions, and yet they are antagonists of one another. How can we explain this?

The explanation of magnesium salt actions seems to reside in the type of the emulsion which it makes in the surface layer of the nerve cells. We ordinarily think of an emulsion as being like the tasteless emulsions of castor oil, droplets or islands of oil surrounded by water; but Bancroft showed, a few years ago, that there are two types of emulsions—the ordinary emulsions of oil in water like the emulsions of butter-fat in milk, and also emulsions of water in oil as is present in ordinary butter. In other words, there are little lakes of water landlocked in oil and with no water channels between them. If we try to pass an electric current through milk it goes through very well. If we try to pass it through butter it encounters a tremendous resistance. Bancroft has shown that when an emulsion of olive oil is made up with sodium oleate, we get emulsions of oil in water; but when we make the emulsions up with calcium soap, we get the exact opposite, emulsions of water in oil. Clowes has shown that in blood and lymph and all the body fluids where we have between 25 and 100 sodium ions for every one of calcium, we get both types of emul-

sion present, and that this balance is necessary for the preservation of most of the phenomena of life.

He also showed that the anesthetic and some of the soporific substances tend to form water-in-oil emulsions, and we have shown that this applies to practically all types of soporific. These oil-in-water emulsions are less permeable to water and the salts and the substances concerned in cell activity, and make a higher resistance to the passage of electricity. This general fact has been shown for living cells by Osterhout.

Last summer, Dr. E. R. Serles and I found that magnesium salts have a much greater tendency to form water-in-oil, the impermeable type of emulsion, than calcium salts have, because the magnesium soap is about three times as soluble in oil as the calcium soap and both are practically insoluble in water. If you add a little

FIG. 17.—Three types of emulsions of water and oil. *A*, Oil in water, the ordinary type of emulsion produced by sodium oleate. *B*, Transition form, met with in serum tissue juices and Ringer's mixtures, in which there is the proper balance between sodium and calcium, showing both types of emulsion existing simultaneously. *C*, Water in oil, produced by excess of calcium or magnesium oleate. (Modified from Clowes, Jour. Phys. Chem., 1916, xx, 407.)

magnesium sulphate to a balanced emulsion made up with Ringer's solution, it changes over to an emulsion of water in oil – what we might call the anesthetic type. So that magnesium salts should tend to coat the surface of the nerve cells over with a less permeable layer, and induce anesthesia, which it does.

If you add a little calcium chloride to such a magnesium oil-water emulsion, it reverses the emulsion; the water droplets run together and you get a return to the normal type of balanced emulsions, and to normal physiological conditions – and, as Meltzer showed, the anesthetized animal wakes up if you inject calcium salts.

We can also explain the analgesic action of the coal-tar antipyretics, and of aspirin and salicylic acid, on the same basis as the action of magnesium salts. All of these drugs dissolve readily in

oil and in the organic solvents, and when this is shaken up with water they give an emulsion of water in oil, just as the anesthetics do. This seems to throw more light upon the manner in which they exert their pain-lessening action than does the theory advanced by Barbour that they act by drawing more water into the blood, perhaps by first increasing the blood-sugar. Barbour's investigations may explain the lowering of temperature by these drugs, but I believe that it is their solubility in lipoids which explains their pain-deadening action.

There is also one point in connection with the clinical administration of the soporific drugs which I would like to emphasize, namely, the effect of the mental state of the patient upon their mode of action. In recent years, many fine soporific drugs have been added to our armamentarium, especially those of the barbital series—veronal, luminal, dial, allonal and amytal, which usually produce a perfectly normal sleep and yet in occasional cases cause delirium, hallucinations and even mania instead. We were interested in seeing whether mental excitement would alter the action of a drug of this series, and Mr. Carl Rice and I studied the question on rats. We found that if a rabbit is put into the same cage with a rat, the rat becomes greatly frightened, although the rabbit will not hurt him in the least. If we gave these rats doses of sodium barbital, which would ordinarily put them into a deep sleep, but kept them frightened by the presence of a rabbit, the drug did not put them to sleep. Even if we gave doses so large that they caused muscular incoördination, the rats did not go to sleep, but still kept trying to climb up the walls of the cage away from the rabbit, though their incoördination was so great that they kept falling back onto the floor of the cage.

This, I think, furnishes a valuable hint for clinical practice. It is chiefly in the nervous and excited individuals that soporific drugs cause delusions, hallucinations and mania. When we go to sleep normally, we put out the light, darken and quiet the room, cover ourselves and warm our skin with the bedclothes and cut down the external stimuli to a minimum; and the drug assists in lowering the intensity of these sense perceptions. But if the internal stimuli of mental excitement keeps on, we have a disorientation from the outside world coupled with an increased flow of internal stimuli, and illusions and hallucinations result.

The corollary to this in clinical practice is that, in order to get

the best effect from a soporific drug, we ought to do everything possible by the ordinary means of nursing and persuasion to bring the patient into a quiet frame of mind before giving the drug, rather than to rely entirely upon the drug to do all the work of inducing sleep.

In conclusion, I would like to say a word to you all as clinicians. In spite of the difficulties that we encounter in obtaining data in the laboratory, we who attempt to teach pharmacology to medical students find ourselves entirely at sea when we try to tell them about exact effects that they may expect to obtain in clinical practice. It is a common saying that all science is quantitative—and where can we find data from which we can tell them how much, how soon and of how long duration will be the effects, even the grossest effects, that are commonly obtained from the most common drugs? In what per cent of the ordinary run of cases will they get any effects at all, and in what per cent will they fail? If there is anything in the world that is of practical importance, it is this question—and yet I have never seen a physician who had ever made the simplest tabulation, even by a +, ++ or 0, as one would tabulate a Wassermann reaction, to record the effects that he himself had observed with any of the ordinary drugs in routine practice. We find in the literature plenty of data upon these effects with digitalis since Mackenzie and Cushny have made such studies rather fashionable for this one drug; one excellent paper by Hanzlik and Scott on salicylates; and some good but conflicting reports upon mercurochrome and tryparsamide. But can anyone give reports on the effects in ten consecutive cases of common drugs like strychnine, nitroglycerine, camphor, belladonna—the relative effectiveness of the various purgatives; the ordinary things that our students should know about under accurately observed conditions? Until we have some such tabulated data, the teaching of pharmacology and therapeutics will continue to be based upon the effects of what are really toxic intravenous doses studied on anesthetized animals, which are quite different, often exactly opposite, from those that are actually obtained with small doses given by mouth or subcutaneously to human beings. How many of us used to believe we got tremendous effects from sixtieths and thirtieths of a grain of strychnine until we began to study the patient's blood-pressure for an hour after giving them and found that actually we got no changes at all or only the momentary effects that were caused by the prick of the hypodermic needle. How many of us are still

giving $\frac{1}{150}$ gr. of atropine and think that we quicken the pulse, though Hugh McGuigan has shown that these small doses usually have the exact opposite effect and render the pulse much slower. In fact, Dr. Nathanson, at the Minneapolis General Hospital, has shown that this is one of the very best ways to cause a slowing of the beat of most rapid human hearts.

A little later we tried out saligenin by mouth in some cases of chronic arthritis at the Minneapolis General Hospital. The physicians were given three sets of capsules, labeled "Special Aspirin A, B, and C." Each set was given for three days consecutively to patients with arthritic pains, the order being varied in different patients. One set contained saligenin, one set aspirin and one set milk sugar. The results made out a fine case for milk sugar in the relief of joint pains. In other words, in a small number of chronic cases, variations in the severity of symptoms are sufficient to account for a great many effects that are often ascribed to drugs; but we can find that out only by objective observations, ruling out the prejudices of the physicians.

Even in the study of a new drug on patients, the personal prejudices *pro* or *con* from which few clinicians are free plays a tremendous rôle. It is very hard to record symptoms objectively. A few years ago I was trying to learn whether saligenin, which is at least five times less toxic than procaine, would be of use in anesthetizing the urethra. One of my friends, an excellent cystoscopist, tried it and reported to me that he got good results, but that he was so fond of alypin that he did not care to make further experiments. I asked him if he would not try a few cases in some of which he would be given alypin, in others saligenin, without himself knowing which he was using, in order to find out whether there was really any difference. He was quite shocked at this suggestion and absolutely refused.

We are living in an era of diagnosis. When we speak of a good clinician, we say he is an excellent diagnostician. Can we not start a little propaganda for learning accurately something about what we are doing to help our patients?

The National Research Council Committee on Medicinal Chemicals is undertaking the problem of coöperative research between chemists and pharmacologists for the study of new substances synthesized in the chemical laboratory. This will do much to put us on a par with Germany in the synthesis of new drugs. But in

Germany the clinicians are willing to try out new drugs and to report upon them—usually only too favorably. Can we not, in this country, establish this coöperation, not only between chemists and pharmacologists but also with university and hospital clinics, to try out new drugs objectively, each on series of patients with control patients untreated or treated by other means, and become independent of Germany? And can we count on you, the highest type of clinicians, for help in that movement?

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DISCUSSION

Dr. George W. Norris: Dr. Hirschfelder has, I think, *assumed* that the laboratory point of view is the desirable and proper one. At the risk of appearing hopelessly reactionary, I must question this assumption. I do this not because I am oblivious to the benefits of the laboratory, which serves me often and well in diagnostic difficulties, but because of what I see among the students of the fourth-year class who are sent to me for instruction in ward-work. These gentlemen shame me almost daily, by my ignorance of chemistry, mathematics and physics, to such a degree that I only with difficulty and with indifferent success cloak my lack of knowledge with an air of respectability. These students of today know a lot about blood chemistry, osmotic pressure, surface tension, the laws of gases, the respiratory quotient and dissociation curves; they know all about the van den Bergh test and the latest modification of the Wassermann reaction. But they do *not* know the visual and acoustic characteristics of the normal chest. They do *not* know the dosage of the twelve drugs necessary in the practice of medicine, nor can they write perscriptions for the same. They do *not* know that the patient needs to be relieved from pain and rendered comfortable, even if an immediate and accurate diagnosis cannot be established. They do *not* know that it is quite as important to recognize the kind of patient the disease has, as the kind of disease the patient has. When I mention to them such measures as hot-water bags, compresses, mustard plasters, massage, cupping or blistering, they seem to regard me as they might an alchemist discoursing upon the philosopher's stone or the transmutation of base metals into gold. They are grievously afflicted with bedside blindness—that condition which Corrigan had in mind when he said that "the trouble with most doctors was not so much that they did not know enough, but that they did not see enough!"

So much for the student of today; but what of the doctor of tomorrow? Quite recently I was asked to see a gentleman who was very ill. He had fever, had been delirious for a week, had cervical rigidity, Kernig's sign, ophthalmoplegia and little else except slight impairment of the right apex and a history of previous sanatorium treatment for tuberculosis. My first thought was tuberculous meningitis, but the family physician could not be convinced that this was not typhoid fever—"Why," said he, "the Widal reaction is positive!" How often it happens that a patient with marked clubbing of the fingers, a chest full of râles and obvious bronchiectasis is bundled off to a sanitarium because some one saw an "acid-fast" bacillus under a microscope! Such instances might be quoted indefinitely and I often wonder—Are not the students getting too much of the laboratory point of view?

Dr. Eugene L. Opie: There can be no fundamental difference of opinion between the clinician and the laboratory worker so long as both are confident that practice must be based on medical science. I feel sure that these young men who have had the good fortune to work with

Dr. Hirschfelder have derived great benefit from the studies which he has described. It has been my experience that only a limited number of medical students can profitably undertake experimental work. Nevertheless, it is desirable that none of those who have the inclination and capacity to profit by research should lack the opportunity to acquire the habit of investigation while they are undergraduate students. It is often said that the technical methods of the laboratory tend to supersede careful clinical study of the patient, and it is doubtless true that novel discoveries seem to have, for a time, more importance than well-established methods. Following the era of investigation into the etiology of disease, immunology, biophysics and biochemistry have come to represent the prevailing trend of research. It is possible that the student of today may fail to realize that clear insight into the nature of disease is dependent upon a knowledge of pathological anatomy. Skill in physical diagnosis, which in its turn has been deduced from pathological anatomy, cannot be replaced by the technical procedures of the laboratory. Furthermore, even from the standpoint of investigation, pathological anatomy and physical diagnosis are not yet exhausted. In the studies of Dr. Rous, experimental methods have been happily applied to problems of pathological anatomy and clinical medicine, and in the investigation of Dr. Hirschfelder and his students problems of physical diagnosis have been subjected to experimental analysis.

Dr. Hirschfelder (closing): I would like at the outset to begin by agreeing entirely with the gentlemen who have seemed to disagree with me. In the first place, I may have created an entire misimpression if I have given the idea that the teaching of pharmacology at the University of Minnesota under my direction is done in this way at all. The routine teaching is not. We give an entirely fool-proof course, as nearly fool-proof as we can, to our students. We have eliminated as far as humanly possible all experimental technic from it, and we have only one blood-pressure done by the students in the whole course. I am entirely under the impression that our students are going to be practitioners of medicine, in which they will not carry around a recording apparatus in their pockets, but will see with their eyes, and therefore we try as far as possible in the laboratory to train them to see with their eyes the things that go on; for example, the dilatation of coronaries after adrenalin and nitrites, and the intussusception which you get when you get intense contractions of the intestines of rabbits under influences which stimulate peristalsis. In many laboratories they set up a lot of apparatus for experiments like this. Our students watch with their eyes and record what they see because they are then coming into their clinical training under men like Dr. Norris who are good clinical observers. If they are blind it is because the eyes they use at bedside are different from what they use in the laboratory. They may get bedside blindness partly from stage fright or bedside fright. As far as the question of proper prescription-writing is concerned, we give them as much prescription-writing as we can find time for in a separate course, and most of our students who go down to the Twin Cities write prescriptions that stand the test. All of them are trained, so that they will not forget it until their

dying day, that when the last physician has given a brown medicine it is their bounden duty to give a red one, and they are all taught how to color it red, and if the last physician gave a red medicine to color their medicine brown. They are also taught how to flavor medicines; because, having graduated from a school where I was taught under a great master how to give kind words and cold water, I occasionally came in competition with men who were much better artists in treatment and who could flavor drugs. I am sure that one sin of omission or commission is not essential. As to whether preparing poultices and compresses and dry cups and all these therapeutic measures should be given in the laboratory, where they never see a patient and where we cannot corral or lasso or drag in the poor unfortunates, or whether they should be given in the wards responsible for their clinical use with very fine nurses and interns to show it to them, that is a question of local expediency. It is an open question whether practical therapeutics should be given in the laboratory or in the ward, and whether it should be given by the professor of pharmacology who has no patients and can only get access to patients if he goes into ward to criticise work of brother physicians, or whether it should be taught by the physician who has patients under control and has them there personally. I believe things should be taught in any school in the place where facilities are most favorably located. So far as I know, these exist only in the medical services. When Dr. Opie properly brought up the question of whether research work by special students takes the place of the bread and meat of the careful routine courses, I would entirely disclaim any desire that that should be the case. The things I grouped together this evening were presented because they happened to fill the frame of one of the subjects that Dr. Beardsley had asked me to talk on. Most of the student researches took a total of sixty-six hours of the student's time. Many of the things encountered in medicine are brand new, and since students have to judge not only the things cut and dried by their teachers and the teachers have digested for them, but also to evaluate new things that are constantly arriving, I think it is perfectly proper, and I will not apologize even to Dr. Opie and Dr. Norris for devoting 1½ per cent of the time of a man's medical education to research.

FOREIGN BODIES IN THE GASTROINTESTINAL TRACT*

By THOMAS A. SHALLOW, M.D.

Did this paper simply add another case to the great number of reported cases of foreign bodies in the gastrointestinal tract, the reading of it would simply waste the time of my auditors.

I wish to emphasize the initial symptoms caused by the ingestion of foreign bodies, to notice how frequent it is that more than one body is present, to follow the course of the object as it descends through the gastrointestinal tract, to point out the complications which may arise, to call attention to the method of isolation at operation and the manipulations necessary in order to permit of the extraction of some of them.

Certain types of people are peculiarly liable to this condition. Paulet,[2] in his learned study, sets forth the causes of swallowing foreign bodies; viz: Instinct, perverted ideas, vicious habits, voluntary introduction, malice, accidental origin, surgical origin. The instinct is universal which leads young infants to carry to their mouths everything they grasp. Dr. J. Chalmers DaCosta, because of this instinct, has defined a baby as "an organized appetite."

In this paper I report three patients under one year of age. The patients were all sent to Dr. Chevalier Jackson's Clinic in the Jefferson Hospital. These cases are instructive. Dr. Jackson points out particularly and emphasizes two facts in relation to foreign bodies in the gastrointestinal tract of infants: (1) The initial symptoms are choking and gagging, which symptoms are manifested by all of the patients in the beginning, though in cases of children they may have been unobserved. Jackson, in his article before the British Medical Association, insists that due attention shall be paid to these phenomena by the physician, that he shall inquire if they have at any time existed and that he shall not

* Read February 3, 1926.

neglect to ask about them even if no symptoms whatever are observed at the time of the medical examination. (2) He further emphasizes, and the record of one of the infant patients in this report illustrates a well-known fact, that is, that more than one foreign body may be and often is present. Jackson[13] insists that our diagnostic obligation is not discharged with the finding of one foreign body by the *x*-rays, but that a ray examination should include the entire anatomy from the nasopharynx to the tuberosities of the ischia in every case.

Case I.—(Dr. Jackson's service.) L. O., male, aged eleven months. He was admitted to the Jefferson Hospital three days after the mother heard the child choking. She at that time discovered that there was a safety-pin missing from her cushion. She consulted a physician who advised her that an *x*-ray examination should be made and that the stools should be watched for the passage of the pin. The *x*-rays disclosed a wide-open safety-pin in the stomach. The child was not apparently suffering any pain; which is the rule. Examination of the abdomen did not disclose any mass or any area of tenderness. The pin was observed by the roentgenologist during twelve days. On the twelfth day it was found that it had passed into the duodenum. Two days later it was still retained in the first portion of the duodenum. It was then decided to remove the pin by operation. Examination the day before the operation showed the pin still in the duodenum.

Operation. Eighteen days after the accident, under ether anesthesia, an incision was made through the right rectus muscle. The duodenum was exposed and on palpation an open safety-pin could be felt in the first portion of the tube. With some difficulty I was able to close the pin without opening the gut. Believing that gastrotomy is a much safer operation than duodenostomy, the closed pin was pushed through the pylorus into the stomach. The stomach was then opened near the pylorus and over the keeper end of the pin by a small incision in the long axis of the viscus. The pin was removed and the stomach was closed by a purse-string suture reinforced by two Lembert sutures. The abdomen was closed without drainage. The convalescence of the patient was uneventful except for a brief period during which there was some elevation of temperature. The temperature rose to 105° F. during the first two days after operation. The child was discharged from the hospital on the twelfth day cured (Fig. 1).

Comments. The period necessary for a foreign body to pass into the rectum is entirely uncertain. If the body is not a sharp one, or a rough one, or an extremely large one, it may be advisable to watch and wait for a time, especially if the body has passed the pylorus. When it has passed the pylorus it has passed the narrowest point of the normal gastrointestinal canal, and unless because of

sharpness, shape or size it should pass successfully through the rest of the canal. There are cases on record of the passage of open safety-pins, but one should not take the chance of such a dangerous delay when the lodged body is sharp. While one is waiting, the feces should be examined daily for the foreign body and also for blood and occult blood. Finding blood or occult blood would be ominous and would signify damage to mucous membrane and per-

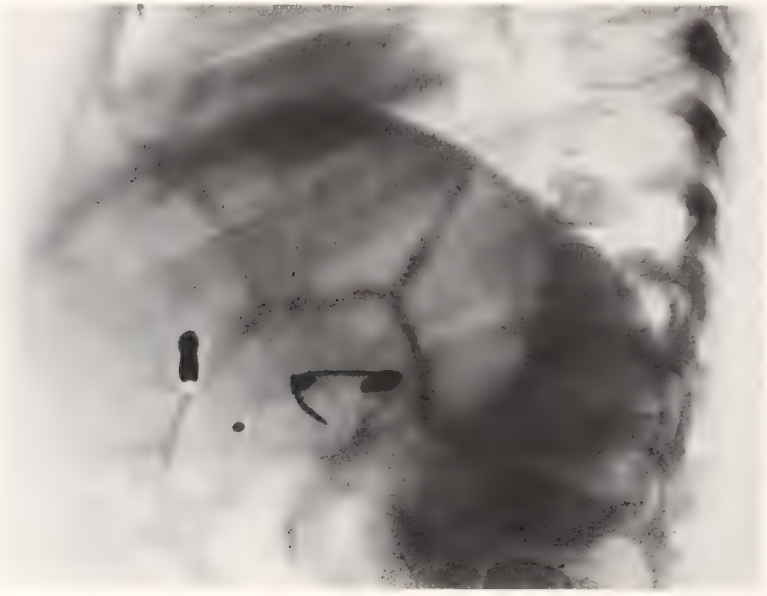

FIG. 1

haps ulceration or danger of perforation. While waiting, daily *x*-ray examinations should be made to see if the body has descended, and every day the abdomen should be palpated to find any area of tenderness or any mass suggesting perforation.

CASE II.—(This case comes under Paulet's classification of "malice"). J. B., aged nine months, was admitted to the Jefferson Hospital, in Dr. Jackson's service, May 20, 1923. Four days before admission the mother noticed that the patient had a slight cough, which was croupy in character. No significance was attached to the cough until two days afterward, when the child vomited a small amount of blood. The family physician was sent for and inquired concerning the possibility of a foreign body having been

swallowed. The mother stated that she could not see how such a thing could have been possible. The following day the child had a severe paroxysm of coughing and choking. The mother put her finger in the child's throat, felt and removed a bent pin. Later, a bent pin, a needle and three cigarette butts were vomited. The same day the bowel movement contained two buttons, one bent hairpin, two closed safety-pins, two bent straight pins and several burnt matches. *X*-ray examination, made by Dr. Manges after the patient's admission to Dr. Jackson's service, disclosed three safety-pins in the pharynx. In the abdomen there was a needle, a straight pin and a slightly bent pin without a head. An esophagoscopy was done on this patient by Dr. Jackson, assisted by Dr. Louis H. Clerf and Dr. Gabriel Tucker. By means of an esophageal speculum a bunch of pins imbedded in the esophagus just below the cricopharyngeal muscle was removed.

This case emphasizes one of the cardinal principles laid down by Dr. Jackson, viz: because you have removed one foreign body, do not conclude that there is not another in the same patient. Look and see. You will remember that the *x*-ray examination disclosed a needle, a straight pin and a slightly bent pin without a head in the abdomen. Subsequent *x*-ray examinations made by Dr. Manges disclosed the needle in the midline of the abdomen, but he was unable to say whether it was in the small intestine or colon. The pins had passed. Daily *x*-ray examinations disclosed the needle to be in the same position after eleven days.

Examination June 17, 1923. Distinct tenderness was detected in the abdomen above the umbilicus and there was some rigidity of the abdominal muscles.

Operation, June 18, 1923. An incision was made a little to the left of the median line The abdominal contents were inspected. There was a mass, which included the termination of the jejunum and the beginning of the ileum, in the center of which the needle could be palpated. The intestinal adhesions were carefully separated. The needle had penetrated from the jejunum into a loop of the ileum, a portion of the needle being in each gut. The needle was removed. No effort was made to close the small openings in the bowel. There was sufficient inflammatory reaction present to insure rapid closure of the holes made by the needle. The abdomen was closed without drainage. The child made an uneventful recovery. The foreign bodies had been introduced by a malicious neighbor (Fig. 2).

Comments. This case shows perforation of two sections of the bowel with the development of a limited peritonitis. The general peritoneal cavity was protected by the adhesions. Such a fortunate event is common but not invariable after needle perforations. This case was not operated upon sooner because of inflammation of the esophagus, which tube had suffered from the lodgment of the foreign bodies. The number of foreign bodies which successfully passed through the gastrointestinal tract is another point of interest.

This case emphasizes the rules laid down by Jackson:[3] "In case of suspected foreign body the ray examination should include the entire anatomy from the nasopharynx to the tuberosities of the ischia." Had this not been done in this case the needle in the intestines would have been undiscovered and would have proven fatal.

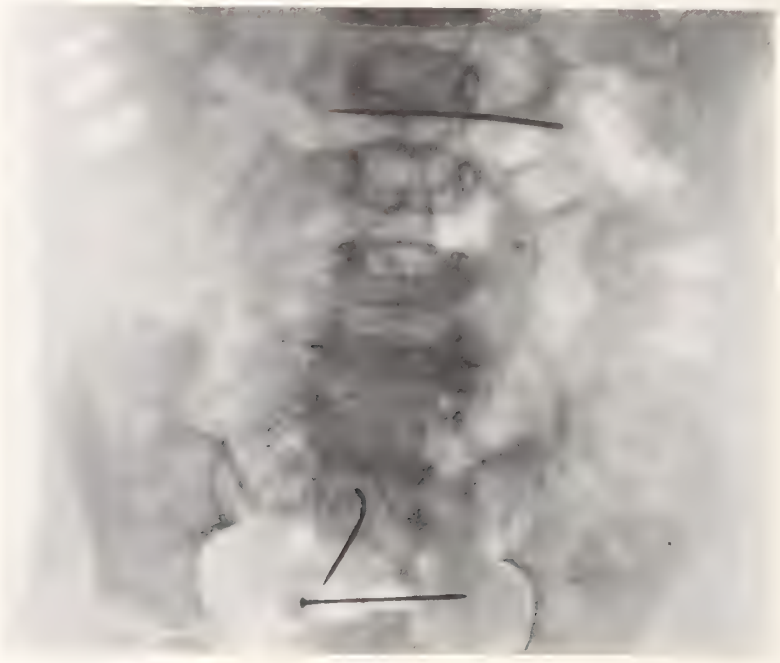

FIG. 2

CASE III.—J. C., aged eight months. The mother's attention was attracted by the choking and gagging of the child. She noticed blood in the mouth. The child was taken to a neighboring hospital where he was examined by the *x*-rays. From this hospital he was sent to Dr. Jackson's service at the Jefferson Hospital. Repeated *x*-ray examinations, made by Dr. Manges, showed a wide-open safety-pin in the duodenum. Operation was performed. An incision was made through the right rectus muscle. A thorough search by palpation of the stomach and duodenum was made without locating the foreign body. The child was immediately taken to the fluoroscopic room. There, with the aid of the fluoroscope used by Dr. Manges, the pin could be seen and we were able to feel it. It seemed to me to be in the third portion of the duodenum, with the point caught in

the wall of the gut. After a great deal of manipulation I was able to push upon the open pin so as to release it from the wall of the third portion of the duodenum and to pass it into the second portion. The second portion of the duodenum was then incised in its long axis and near its convexity, after the duodenum had been partially mobilized. The open pin was removed. The duodenal opening was closed by a purse-string suture reinforced with Lembert sutures and the abdomen was closed without drainage.

Following the operation there was considerable shock, but reaction was rapid. The child made a complete recovery and is now well (Fig. 3).

FIG. 3

Comments. This case teaches us the absolute necessity of close coöperation between a skilled roentgenologist and a surgeon, so that we may have the advantage of the use of the fluoroscope for the localization of foreign bodies during operation. This procedure reduces the time and hence the shock of the operation, enables us to find a metal body and thus not only preserves the patient's life but saves the surgeon the chagrin of operative failure.

CASE IV.—Case IV is classified by the plan of Paulet as one due to "perverted ideas." H. L., female, aged thirty-six years, a lunatic. This

patient, while an inmate of a hospital for the insane, swallowed a number of foreign bodies. During the process of swallowing a safety-pin, choking and gagging were observed by a nurse. She was sent from a neighboring state to Dr. Jackson's Clinic. *X*-ray examination disclosed an open safety-pin in the esophagus. An open safety-pin, a common pin and a straight pin without a head were seen in the abdomen. The open safety-pin was

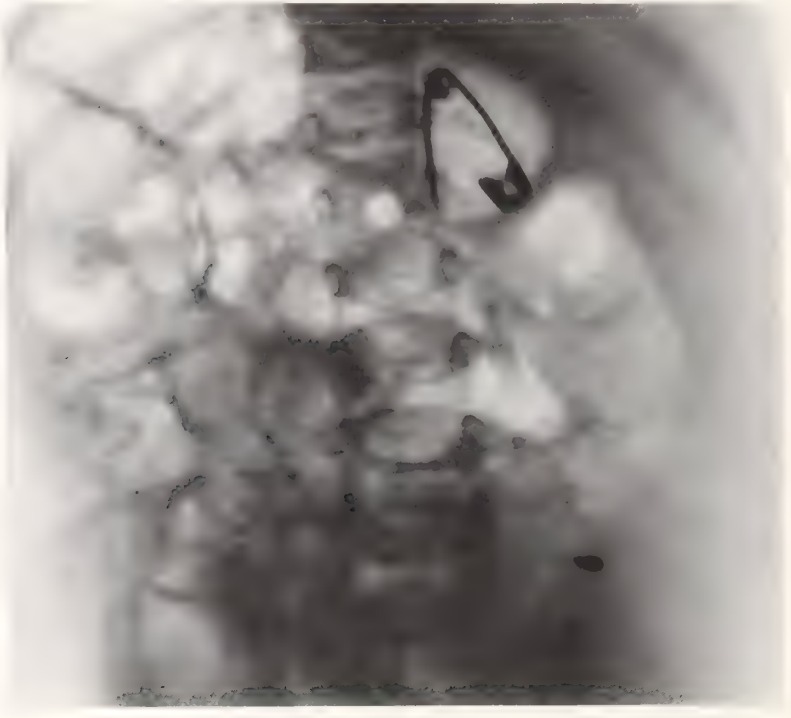

FIG. 4

removed from the esophagus, by peroral esophagoscopy, by Dr. Gabriel Tucker assisted by Dr. Louis H. Clerf. The abdominal foreign bodies were observed during three weeks by means of the *x*-ray and the fluoroscope. Twelve days after admission the open safety-pin was passed per anus. The two straight pins progressed to the large bowel. At the end of the twenty-second day after admission to the hospital the *x*-ray disclosed the pins still in the cecum. It was evident that they were not progressing. Operation was recommended.

Operation. A lower right rectus incision was made. The pins were found to be imbedded in the ascending colon, but as yet neither pin had

penetrated the peritoneal coat. The pins were pushed through the wall of the bowel and removed. The openings were closed as before described. The patient made an uneventful recovery.

Comments. This is another case of multiple foreign bodies in which a potentially fatal foreign body would have been overlooked had not the above-mentioned rule (Case II) or ray examination[3] been followed. In this adult case an open safety-pin passed through the intestinal tract. In the infant cases reported in this paper the open safety-pins were lodged. There is always a great probability

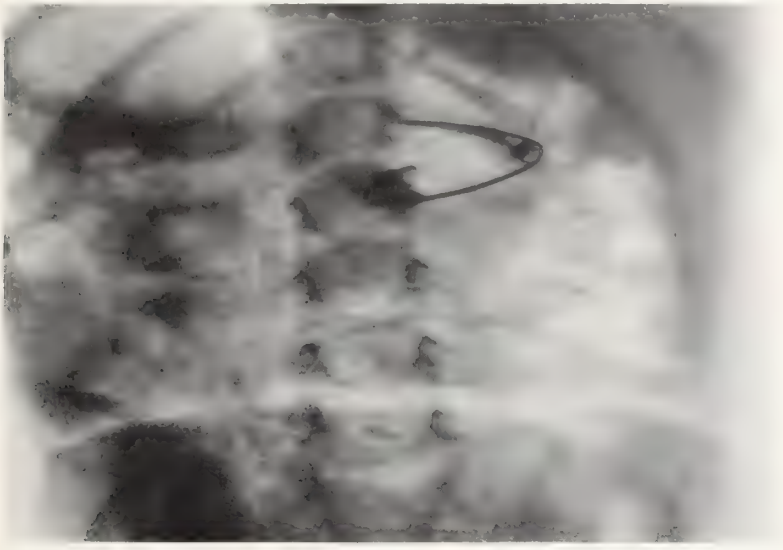

FIG. 5

that a straight pin, an open safety-pin or a needle will perforate the bowel wall in an adult as well as in a child.

The persons among the insane particularly prone to swallow strange foreign bodies are dements. Delusional lunatics occasionally swallow them. Some lunatics take them with suicidal intent. Idiots and imbeciles and hysterical persons may also swallow foreign bodies. Constant watchfulness is necessary on the part of attendants and any unusual conduct or any suggestive symptom should be reported to the physician at once.

Case V.—H. L., male, aged fifty-six years. Was admitted to the Jefferson Hospital for a growth involving the right tonsil. During the course of treatment (under local anesthesia) by means of radium needles, it was discovered that one needle containing 12½ mg. of radium was missing. Each needle was threaded with heavy silk as a precaution against swallowing the body. In spite of this an *x*-ray examination, made by Dr. Manges, disclosed the needle in the abdomen a little to the left of the median line and apparently in the small intestine. At the end of twenty-four hours the

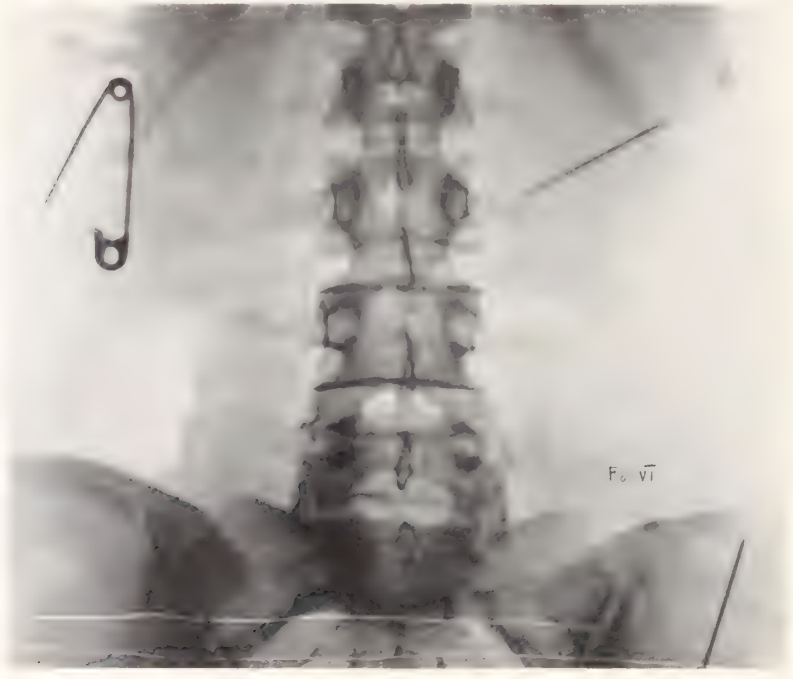

Fig. 6

needle, as observed by the *x*-rays, was in the same position as on the preceding day. Operation was recommended because of the probable effect of the radium on the tissues of the gastrointestinal tract.

Operation. An incision was made at the outer edge of the left rectus muscle (under local anesthesia). A careful search of the small intestine failed to reveal the needle. The patient was taken to the fluoroscopic room. There, with the aid of the fluoroscope used by Dr. Manges, I was able to seen the needle just below the diaphragm. With some difficulty the needle was grasped and proved to be in the stomach. The stomach was incised on the anterior surface over the eye of the needle. The needle was removed. The incision in the stomach was closed with a purse-string suture

and the abdomen was closed without drainage. The patient made an uneventful recovery from the abdominal operation and was discharged from the hospital in three weeks. There were no apparent ill-effects from the radium which had been swallowed.

Comments. The question arises, would this patient have swallowed the radium needle had he been under a general anesthetic? We have observed the extreme irritability of the throat during operative procedure under local anesthesia and the constant swallowing attendant on any prolonged manipulation of the fauces and pharynx. This case also exemplifies the necessity of fluoroscopic coöperation in certain cases during the surgical procedure.

Conclusions. 1. Needles, safety-pins and straight pins in the food passages of children should be removed early to anticipate the danger of perforation. When the child's condition does not warrant surgical intervention (for instance, as in the case of esophagitis, previously cited, or when a foreign body in the air passages calls for immediate attention), a daily study of the stools should be made to determine the presence of macroscopic blood or of occult blood. If blood is found it is significant of definite traumatism to the mucous membrane or beginning ulceration.

2. Foreign bodies, even open safety-pins, progress by rotating on their long axes, as is exemplified by Figs. 3, 4 and 5.

3. The surgeon should not attempt to remove a foreign body from the food passage without the coöperation and the assistance of a roentgenologist during the course of the operation, in order that the fluoroscope may be used at once, when necessary.

4. Systematic *x*-ray examinations must be made daily, if we elect to delay, in order to eliminate the danger of overlooking another body or other foreign bodies.

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DISCUSSION

Dr. Astley P. C. Ashhurst: Such a paper as this recalls, first of all, to my mind a tale we were told of old Dr. Nathaniel Chapman, who was a famous wit. Dr. Nathaniel Chapman was consulted by an anxious mother, who said the baby had swallowed a coin. Dr. Chapman refused to get

excited and said, "Are you sure it wasn't a counterfeit?" and being assured it was not, said, "Then it will pass!"

Now I do not see how this paper can be discussed except in the narration of personal experiences. During the World War many of us had a very large experience in the removal of foreign bodies from unusual locations. Two of the cases struck me as so interesting that they impressed me particularly. The first soldier had a compound fracture of the femur, not a particularly large surface wound, and when the thigh was opened by *débridement*, I pulled out what I thought at first was a large, loose bone-fragment, but it was the timing-cap of a shell! This, as many of you know, resembles the combination lock on a modern burglar-proof safe. This timing-cap and the attached end of the shell were inside this man's thigh. Another patient had a perforating wound of the knee-joint; in cleaning out the knee-joint I picked out two leaves of a tree, which were easily recognized as beech-leaves. I asked the man about it the next day and he said he was in a beech forest when struck.

Apart from war surgery, we often see retained foreign bodies in wounds occurring in civil surgery. A patient, years ago, came into the surgical dispensary at the Episcopal Hospital with a sore arm, red and inflamed, and with a couple of old scars; he said that twenty-five years before he had been cut by glass, and he thought the glass was trying to come out now; I said that was nonsense; but when I cut open the arm, there was the glass trying to come out. Another instance of glass was this: a girl, thirteen or fourteen years old (1914), cut her hand with glass about ten years before I saw her. Ever since that time she had had hyperesthesia and some atrophy in the distribution of the median nerve. She cut the same hand again with glass about a year before I saw her, and the pain in the distribution of the median nerve grew worse. An *x*-ray showed a fragment of glass, and though at operation I looked everywhere for the glass I could not find it; after looking everywhere else, I opened a small bulb in the median nerve under the annular ligament and out of it popped the glass, and I have the piece of glass here tonight and shall be glad to present it to the Mütter Museum.

Among the dangerous things which are swallowed, which Dr. Shallow did not mention, are fish-bones. A man about sixty-two years of age (1916), living in Delaware, while in perfect health, all of a sudden had a chill; the next day another chill and the third day two chills, and continued having one or two chills every day for some weeks; he seemed to be sick in no other way, but rapidly lost weight. His physician consulted Dr. William Egbert Robertson, who went to see the patient and put him under my care in the Episcopal Hospital. He was very septic and sick, and was not jaundiced but there was tenderness over the liver, and Dr. Robertson's diagnosis was abscess of the liver. He did not know what would cause him to have abscess of the liver. I explored by the pleural route from behind. Having found nothing, and the man getting no better, after a couple of days I explored again through the abdomen. He was a fat man and I could feel with my fingers, but I could not see with my eyes, somewhere in the region between the duodenum and the under surface of the liver, where there were adhesions, a calcareous body which after some difficulty I managed to

extract partially from the duodenum and partially from the liver. It was a fish-bone. The man died without being relieved, and autopsy showed that he had abscesses all through his liver.

About three years ago, a man, aged seventy years, came into the hospital with a mass in his right iliac fossa, a history of being sick at home for weeks with emaciation and fever; and I thought he had an abscess from perforation of a carcinoma of his cecum. I operated on him and on opening the abdomen found pus, and in the bottom of the abscess near the cecum I found a fishbone. The appendix was normal. That man got well.

I once took a hat-pin out of a man's bladder; the man thought that he had an obstruction of his urethra; he said he pushed the hat-pin in head first, so it would not hurt him, and as he saw it disappear he got excited and broke the point off and could not recover the remains, 5 or 6 inches long. I removed it by suprapubic cystotomy and he made an uneventful recovery.

A girl, aged four years, had trouble passing her urine; her mother took the child to the family physician (Dr. Bigley), who treated her for cystitis for several weeks. Finally, as she grew no better, he put a sound in the bladder, felt a stone, and sent her to the Episcopal Hospital, where an *x*-ray was made which I have here to show you, Mr. President and Fellows. The *x*-ray showed a good-sized safety-pin inside the bladder, inside the stone. Dr. Haines yesterday tried to extract the stone by the cystoscope, but he was unable to get it out. This morning I removed the stone suprapubically and have it to show you tonight.

Dr. Shallow mentioned in passing, without dwelling upon it too much, foreign bodies that surgeons may leave inside patients—gauze sponges, for instance. When I was working in the dispensary, a woman attended for some weeks after an abdominal operation, with vague abdominal complaints. After a considerable absence she returned, bringing with her a long sponge which she said she had passed from her rectum. Her abdominal but not her mental distress was relieved. This is known to have occurred in other instances. It occurred in Base Hospital 34, France, after an abdominal operation; after some weeks of misery a sponge was passed from the rectum. The case was reported by Dr. John P. Jones (1920). I once operated on a patient with an appendiceal abscess; he was pretty sick and the abscess extended up on the outer side of the ascending colon. It was then my habit, as in many hospitals, to have sponges counted before use and after, and the nurses are held responsible for a correct count. The report was made that the sponges were correct and I sewed up the man's wound, with drainage. He did well for about a week, when there was a sudden rise of temperature and a tender mass developed in the flank, which I thought was an abscess. Under local anesthesia I made an incision and as soon as I opened the parietal peritoneum there was a gush of pus, followed by the end of a gauze sponge. I pulled it out without the patient knowing it, and he made an uneventful recovery. On another occasion the sponges were counted and one was reported missing. I was quite confident it was not inside the patient, and I closed the wound. The next day the nurses reported they had found the missing sponge when cleaning

up the operating room. Since then I never have had them counted, and if I have left any inside neither the patient nor any body else has known it.

I have one more patient to refer to, who was Dr. Gwilym Davis'. He never reported this case because he was opposed to reporting sensational cases. I do not think he would mind my doing so, however, on this occasion. A Polish man came (1909) to the Episcopal Hospital Dispensary. He could not speak English, but made motions that something was wrong with his throat; he was given a cough medicine. The next day he returned, feeling no better. More particular inquiry indicated he had swallowed something which hurt his throat. He was then sent to the receiving ward, where an attempt was made to pass an esophageal bougie. As this met an obstruction, *x*-rays were made. This was seventeen years ago, when the *x*-ray was not as accurate as it is today, but it showed something in the esophagus, apparently a fork. Dr. Davis did external esophagotomy and after much trouble extracted a fork, which was tightly wedged inside, its prongs having been carefully wrapped up by the patient in a ball of twine for fear they might give him pain as he swallowed the fork. The patient's family stood beside him, as he came out from under the anesthetic, and the first thing he said was, "How many did they find?" A reëxamination of the *x*-ray showed some mysterious shadow in the region of the stomach which looked like an egg-beater; so after a few days Dr. Davis opened his stomach and removed another fork and three spoons. The man eventually died from perforation of his stomach in an area wounded by the utensils during their sojourn in the stomach.

Dr. Hubley R. Owen: About a year ago I had in the Jefferson Hospital a boy who swallowed a piece of glass broken from the top of a marmalade jar. His mother made a marmalade sandwich for him and as he swallowed he had a sharp, excruciating pain in his throat. I saw the patient within an hour and made a diagnosis of foreign body in the throat, because of the symptoms presented and because of the fact that there was a piece of the top of the marmalade jar missing. The boy was sent to the Jefferson Hospital. Dr. Chevalier Jackson saw the patient in consultation the following day and corroborated the diagnosis, and stated it was impractical to remove the glass by means of an esophagoscope because the glass had penetrated the walls of the esophagus. An *x*-ray picture taken by Dr. Manges showed the foreign body. The case was treated tentatively for a number of days. He finally developed a retroesophageal abscess. This was opened through a lateral incision. The pus was evacuated from the abscess and the piece of glass removed. The boy made an uneventful recovery. Several months ago I had as a patient a member of the police department who received a gunshot wound of the popliteal space. An *x*-ray showed a fragment of bullet lodged in the popliteal space. I did not think this small fragment would give him any trouble and did not advise its immediate removal. He reported several weeks later, complaining of a dull, constant, aching pain in the popliteal space. I then decided to remove the foreign body. The attempt was made in the fluoroscopic department of the Philadelphia General Hospital. I made an incision in the popliteal space and luckily

introduced my finger into the wound to see if I could feel the foreign body, but instead of feeling a foreign body I felt an aneurysm. Further examination showed that this was an arteriovenous aneurysm. This arteriovenous aneurysm was operated upon but the foreign body was left *in situ*. This patrolman made an uneventful recovery from his arteriovenous aneurysm, but I have never removed the foreign body. He has had no symptoms relative to the presence of the foreign body. I had three similar cases while working in the Base Hospital 38 abroad: two dissecting aneurysms of the lower extremity and one dissecting aneurysm of the thigh, resulting from machine-gun bullets. I mention these cases to impress the fact that one should keep constantly in mind the possibility of an aneurysm following gunshot wounds.

Dr. Ashhurst recalled one patient from whom he removed a piece of glass imbedded in his arm for twenty-five years. I had recently a similar case. A patrolman reported to me, complaining of pain in his right forearm. A thorough examination, including an *x*-ray, did not divulge the cause of his pain. There was, however, a very indefinite area of induration in the interosseous space. A number of weeks later, with some reluctance, I made an incision over the indurated area and removed a triangular piece of glass. There was no scar on his entire right upper extremity which could have been a wound of entrance for this glass. The only wound he had was on his left shoulder. He stated that, some years ago, prior to his appointment in the police department he was a bartender and was cut on his left shoulder by a broken tumbler while in a fight in the barroom. Of course, we hear of needles traveling to parts of the body far distant from their wound of entrance, but it is difficult to conceive of this piece of glass having traveled from the left shoulder to the interosseous space of his right forearm.

The importance of the use of the fluoroscope in the removal of foreign bodies has been mentioned by Dr. Shallow. One should never attempt to remove a foreign body without access to the fluoroscope: we are thoroughly familiar with the difficulties of removing foreign bodies such as needles, etc., which may appear to be readily accessible but prove most difficult to remove.

Dr. Willis F. Manges: I shall limit my few remarks to the foreign bodies of the esophagus and gastrointestinal tract. Foreign bodies lodge in various portions of the gastrointestinal tract either because of their size or their shape or length or because of some sharp or pointed projection. The large, obstructing foreign body is the common type in children and is very rare in adults, whereas the small splinterlike foreign body is common in adults and rather rare in children. The majority of the large foreign bodies lodge at the level of the suprasternal notch because that is the first narrow point in the esophagus. However, they may lodge in any other portion of the esophagus or in any portion of the gastrointestinal tract if there is some abnormal narrowing of the lumen or sharp curves because of adhesions. The small, sharply-pointed foreign bodies are also apt to lodge fairly high in the esophagus, but they occasionally do reach into the lower half. These foreign bodies lodge because some sharp projection or one end becomes

partly embedded in the walls of the esophagus. They do not interfere with the passage of liquids and sometimes do not interfere with the passage of semisolid foods. Occasionally, one of this type of foreign body may penetrate and pass through the esophagus into the periesophageal tissues. The piece of glass reported by Dr. Owen is one of this type. I believe that this foreign body passed entirely through the esophagus into the retroesophageal space at the time of the accident, that is, it did not stay in the esophagus at any time. Shortly after this accident the patient was studied by an extremely careful roentgenologist and it gave none of the usual signs of foreign body, nor was the swallowing function interfered with. I saw the child only after infection had started in, and a great deal of swelling pushed the esophagus and trachea forward so I could not help but see that there was a lesion present. There was a bubble of gas present in the tissues of the neck. Films made at this time showed the small fragment of glass lying in an anteroposterior direction in the deep tissues of the neck. I believe that when it was examined by means of the rays before it was really hidden by the bones of the neck.

I believe that there is an element of spasm controlling the lodging of foreign bodies. For instance, small coins may lodge, whereas coins of larger size in patients of the same size or age may pass. Coins even as large as a half dollar have passed throughout the entire gastrointestinal tract of a child, and we have frequently seen pennies lodge in the upper end of the esophagus and stay there for weeks or until they were removed. At present there is a child in the hospital with a fifty-cent piece several inches above the diaphragm in the esophagus. It has been there for five years and has ulcerated through the esophagus, through the pleura and lung and into the left bronchus, so that we could see bismuth mixture pass from the esophagus into the left lower lobe bronchus during the act of swallowing.

This brings to mind a point with regard to the diagnosis. It is remarkable how many physicians do not pay attention to the history that is given them by the patient. In the case of this little boy, the father has insisted throughout the five years that the boy had swallowed a half dollar. None of the physicians would believe him because they said it was impossible for a child five years of age to swallow anything so large as a fifty-cent piece. Some years ago, an elderly gentleman came to the city to the late Dr. Kyle because of obstruction in his esophagus. He could swallow only small quantities of liquid and had had trouble for seventeen years. At the time of the accident he begged a surgeon to operate to remove his false-tooth plate from his esophagus, but the surgeon refused, saying that his false-tooth plate would probably be found somewhere else. This man went for seventeen years with his false-tooth plate in his esophagus. It was then taken out by Dr. Kyle and Dr. Lewis with the aid of an esophagoscope and without a surgical operation.

A foreign body of considerable length may be unable to get around an angulation in the gastrointestinal tract. I once saw a patient who swallowed an ordinary wooden leadpencil, full length—in fact, it had never been sharpened. It went as far as the cecum and apparently stopped there because it could not get the right direction through the ileocecal valve.

Dr. Nassau removed this pencil, and at the time of operation there were several small gangrenous spots in the intestines. I once supposed that a jackstone would not pass because of its marked irregularity, and I supposed open safety pins would not, but they do. We have seen many open safety pins go through, even in small children. In one case we watched a closed safety pin for about thirty days. It lodged at the duodenojejunal junction. The mother refused operation and finally took the patient to another institution for surgical removal. We feel that if a foreign body remains in any one location in the gastrointestinal tract beyond the pyloric sphincter for more than a few days that surgical interference should be considered. On the other hand, so long as there is any change in position from day to day, there is apparently no harm in waiting. The roentgenologist should not assume the responsibility for treatment, that is, he should not say that this patient should or should not be operated or that one should wait for a longer time before operating. But if the patient does come to surgical operation, then I think that the operation should be done when a fluoroscope is available to guide the surgeon to the foreign body without undue manipulation. Needles, for instance, are very apt to lodge in the intestines, because both ends are rather sharp, and they are extremely difficult to find by palpation. Other foreign bodies may change position during or just before operation. For foreign bodies in the abdomen, we have not attempted the use of right-angled fluoroscopy because a small foreign body, such as a needle, can hardly be seen in the lateral direction. One should resort to the so-called "near-point" method with single *x*-ray tube and screen. One puts a blunt instrument of the forceps variety into the abdomen and with the aid of the fluoroscope guides it to such a point where it will readily disturb the foreign body. The surgeon then may either open the forceps and gently grasp any tissue that is in relation to his forceps, or, with the fluoroscopic screen removed, then palpate in the location so determined. This method of localization was very popular during the late war, not only because it was a satisfactory method of localizing the foreign body with relation to the nearest point on the surface, but because it was possible to localize a large number of foreign bodies in a short period of time. When the foreign body is in the chest, of course, one should use a right-angled fluoroscope.

Dr. John H. Gibbon: It is very important to locate a foreign body by plate or flouroscope just before attempting its removal. We have all had the chagrining experience of searching for a foreign body which had changed its position between the time of its localization and the operation. Many years ago I opened a small child's abdomen with the object of removing a coin from the stomach. It had remained in the stomach for about two weeks, the last plate being made a day or two before operation. I could not feel the coin in the stomach or intestine and was on the point of examining the child under the flouroscope when I introduced a finger into the rectum and removed the coin.

URETERAL STRICTURE, A NEW ABDOMINAL SYNDROME: ITS ETIOLOGY, DIAGNOSIS, PATHOLOGY AND TREATMENT*

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(By Invitation)

Skepticism and the adoption of a new idea only after a reasonable demonstration of its practical value are signs of a healthy attitude in medical practice. Rejection of a theory because of its novelty or because its acceptance leads to the overthrow of traditional views and practice may indicate a state of smugness or self-complacency which in some fields may be condoned without argument, but which in medicine may cause serious injury to the patient who has entrusted the physician with the care of his health.

Although Fitz, of Boston, published his epoch-making paper on appendicitis in 1886, some of us are old enough to remember many shocking disasters occurring in the early years of this century because in the minds of some medical men acute appendicitis was still a novel idea and looked upon almost as an obsession of the surgeon. Indeed, it was only when the layman had experienced some of these disasters in his own or in his neighbor's family, and had thus learned to suspect acute appendicitis and to carry his case direct to the surgeon, that some of the more conservative members of our profession awakened and added this disease to their daily reckoning with acute abdominal disorders. But although, fortunately, the disease whose syndrome we are discussing tonight rarely leads to such immediate disasters as so often occur in cases of acute appendicitis, it is certain that ureteral stricture is an abdominal disease of sufficient importance to warrant our making some comparisons between the two conditions.

As a gynecologist I naturally have far fewer appendicitis patients referred for diagnosis and treatment than the general surgeon, and I am not in a position to judge from personal experience regarding

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the relative incidence of this disease. As yet our hospital records are not helpful in comparing the incidence of ureteral stricture with that of appendicitis, because in most hospitals ureteral stricture is not being systematically diagnosed.

Because of the early diagnosis and surgical treatment of most cases of appendicitis, and because of the neglect of proper diagnosis in most patients suffering with ureteral stricture, one is justified in stating that today ureteral stricture as a source of incapacity and morbidity ranks equally with or perhaps above appendicitis. Ureteral stricture as treated, or rather as neglected, today is not only responsible for the physical discomfort and incapacity which it directly causes, but must also be charged with the results of mistaken diagnoses. To make this plain I need present only a partial list of the evil consequences following our neglect of this disease. On the economic side there is tremendous loss in disability, time, and hospital expense incurred in following various lines of treatment founded on erroneous diagnosis. When such errors lead to multiple abdominal operations, the patient not only suffers the undermining of the nervous system consequent on the operations and repeated disappointments in obtaining results, but she is likely to acquire permanent morbid processes as a result of the operations. And, finally, if we need further presentments to make the indictment impressive, let us add the last and most important count, namely, the resultant damage to the kidney when we fail to make an early diagnosis of ureteral stricture. In the face of these indisputable facts, then, can the members of our profession in these days of preventive medicine still continue to contemplate these mistakes with equanimity?

My sponsor for this meeting, Dr. Beardsley, has asked me to emphasize the viewpoints on etiology and symptomatology of this disease for the benefit of the busy general practitioner in order that he may bear in mind the possibility of its existence in doubtful cases.

At the risk of making my paper seem elementary after all that has been written on the subject, I shall try to present a brief summary of these features of the disease founded on an experience with more than 2500 cases studied in the past fifteen years.

Definition. Ureteral stricture is an intrinsic disease of the ureteral wall resulting in narrowing of the lumen, which leads to varying degrees of stasis in the urinary stream.

Etiology. This narrowing may be congenital or acquired. The congenital types probably most often represent anomalies of development, but I believe that time and more careful study will eventually prove that some of the congenital cases are caused by inflammatory processes derived through the placental circulation. By far the greater number of ureteral strictures, however, are acquired. We see an occasional stricture of traumatic origin either from a severe accident, a gunshot or stab wound, or from injury at operation or childbirth. Syphilis may be responsible for an occasional case. I have seen one stricture located at the pelvic brim which was apparently secondary to the pressure and inflammatory action of an adherent ovarian cyst. I have seen pressure by uterine fibroids cause hydronephrosis and hydroureter above, but apparently without causing intrinsic disease of the ureteral wall. Adenomyoma as it spreads across the pelvis sometimes involves the ureter, causing stasis from encircling pressure. I have never seen a stricture which could be ascribed to the juxtaposition of a pus tube. A fairly large group of ureteral strictures are associated with cancer of the cervix. The inflammatory process in these cases may involve the periureteral tissues more than the ureteral wall itself and may be present at the time of operation, or it may follow the inevitable trauma and infection due to operation. In a new group of which I have seen several examples the periureteral and ureteral infiltration in the broad ligament region has apparently followed the use of the cautery or radium, before, during or after the operation. In renal tuberculosis disease of the ureter is often a secondary manifestation.

As a gynecologist I have been constantly expecting to see stricture in the broad ligament region which could be ascribed to inflammatory disease of the cervix, but thus far I have not been able to trace this connection.

Simple inflammatory stricture due to a focal infection in some other part of the body is so overwhelmingly frequent, when compared to stricture from all other causes, that we are justified in saying that the disease usually originates from some such focus. The acute infectious diseases of childhood probably leave inflammatory areas in the ureter, the results of which may not be apparent until later in life. Any localized acute or chronic inflammatory process may finally lead to ureteral stricture. Carbuncle, onychia, intestinal, gall-bladder, appendix, and bone inflammations probably each account for a certain percentage of stricture cases; but

my experience seems to point to the infections about the head as causing by far the largest number of inflammatory conditions in the ureteral wall. My reasons for this view are briefly as follows: (1) A careful history often reveals that the symptoms due to stricture began soon after an attack of tonsillitis, sinusitis, or a dental abscess. (2) Experience has taught me that the treatment of the stricture area is often futile until one has located and eradicated infection in one of these areas. (3) Dilatation of the stricture area may result in cessation of all symptoms, and in the clearing up of a pyelitis, if present, only to have the patient come back weeks, months, or years later because of the return of all the symptoms following a fresh attack of sinusitis, tonsillitis, or dental inflammation, or after a visit to the dentist. (4) After a tonsillectomy on a patient who has had treatment for stricture we occasionally have the patient complain far more of pain in the ureteral and renal regions than of the local discomforts from the operation. A cystoscopy a few days after the tonsillectomy shows, in some cases with such ureteral reaction, a wide area of edema and redness surrounding the ureteral orifice, in contrast to the normal or approximately normal appearance of the mucosa in all previous examinations. (5) During and after the grippe epidemic of 1918, many of my "cured" stricture patients returned for further treatment after a severe attack of grippe. (6) Another reason for considering ureteral stricture as originating from a focal infection is the fact that it practically always occurs bilaterally. From the fact that most strictures occur in the broad ligament region from 2 to 5 cm. above the bladder, or in the iliac gland region from 3 to 5 cm. below the pelvic brim, I have advanced the theory that the systemic infection or toxin probably first attacks the lymphatic glands in these regions and later invades the neighboring ureteral wall.

Symtomatology and Diagnosis. Because our attention has but recently been called to this disease, the general practitioner is likely to consider that it is of rare occurrence and that its symptoms are so vague as to make a diagnosis difficult and available only to the specialist. As a matter of experience the physician who keeps this disease in mind, when considering any vague abdominal disorder, is surprised at the frequency of its occurrence, and at the ease with which a fairly certain diagnosis is made without the aid of the urologist.

The urologist who is treating ureteral stricture constantly marvels at the number of patients who come to him on the recommendation of other patients, whom he has relieved, and who recognize the symptoms in their friends and send them for treatment over the head of the family physician, who has failed to make a diagnosis, thus repeating the history of what happened in the early days of appendicitis work.

You would take offense if I were even to hint that you are incapable of making a reasonably certain diagnosis in most cases of chronic gall-bladder disease or appendicitis without calling a specialist to help you; but careful anamnesis and physical examination in a case of ureteral stricture usually furnish far more clues for an accurate diagnosis than are obtainable in the above two diseases with which you are so familiar. There is probably not a man in this audience, with a reasonably large practice, who has not one or more of these patients on his visiting list, patients whom for the most part you have consigned to your group of neurasthenics supposedly in the enjoyment of miserable health. Some of these patients are decorated with a half dozen abdominal scars, mute or painful testimony to the zeal of your surgical and gynecological brethren, who have been trying to aid you in making a diagnosis. Others have tried the patience and skill of your gastrological confrères and are still complaining of the vague abdominal pains, the accumulations of gas, or perchance a continued mucous colitis. Others are wearing the harness of your orthopedic colleagues in a fruitless effort to relieve the back, hip and thigh pains. Possibly you have under you care one of the rare individuals who has been sentenced to one or more terms at Saranac, Asheville, or Colorado, in spite of the fact that the most exhaustive clinical methods have failed to reveal signs of tuberculosis, and your patient still complains of a vague indefinite backache located higher than the usual kidney ache, a slight daily rise of temperature, and general malaise and asthenia.

If you so will, you can learn tomorrow, almost to a certainty, and without the help of a urologist, whether one of these trying patients under your care has ureteral stricture as the physical foundation for the so-called neurasthenic condition.

Careful anamnesis elicits in about 75 per cent of these cases a history of bladder trouble. This may be so slight that your patient has not mentioned it, and on the direct question may answer that

she has no bladder symptoms, only a slight frequency when she gets nervous or excited, or a frequency at the time of the menstrual period, or in association with a "cold" or sore throat, or with an arthritic attack. From this almost negligible symptom of intermittent frequency we see all grades of bladder distress up to the condition of complete incontinence of urine in patients in whom we find no other lesion of the urinary tract than a ureteritis, and for whom we obtain perfect relief by dilatations of the ureteral stricture alone or combined with attention to some area of focal infection if this has not already been treated.

Urinalysis may be most helpful in the diagnosis; sometimes, however, it may be only slightly suggestive, or at times most misleading. In 20 per cent of your stricture patients there is a chronic pyelitis on one or both sides, and the urine will give characteristic findings. In 50 per cent you will find only a few erythrocytes, a few leukocytes, albumin from a slight trace to a large quantity, or casts, or a combination of these elements. Too often in the past such evidences of disease, when found in meager quantity and only on the most painstaking urinalysis, have been considered as of no significance. In the remaining 30 per cent of stricture cases the urinalysis is completely negative and patients in this group may present symptoms strongly pointing to the urinary tract and yet be sent to the gastroenterologist, the orthopedist, or the exploratory laparotomist for a diagnosis simply because of the negative urinalysis.

Finally, as a general practitioner, you do not need the urologist to aid you in making a careful physical examination. In the stricture patient, as in any other, you may or may not be able to palpate the kidneys. In women you can usually palpate the right kidney. The kidney above a stricture is usually, but not always, tender. Even when the kidney cannot be felt, you will usually elicit tenderness in the upper flank.

Palpation of the ureter where it crosses the pelvic brim practically always elicits tenderness in stricture patients. Frequently the patient will refer the discomfort so caused to the epigastrium and complain of nausea, or "that old gas pain;" or the discomfort is referred down the ureter to the "ovaries" or to the bladder with a desire to void.

Palpation of the ureter in the broad ligament region usually distinguishes the area of greatest tenderness. The pain may be referred to any portion of the upper ureter, and even to the kidney.

The patient often volunteers: "That is the old bladder pressure" or "That is the pressure on the bowel," or "That is my menstrual pain," or "That is the pain I have when using the douche nozzle," or "That is the pain I have with the sexual act." Dyspareunia in the majority of cases is due to the presence of ureteral stricture, and "ovarian neuralgia" is probably more often a ureteral than an ovarian pain. When a patient complains of "falling of the womb" and you find on taking her history and examining her that she is a nullipara, or that she has an outlet in an excellent state of preservation, and apparently normal internal genitalia in normally high position, do not fail to palpate the ureters for the source of her symptoms, for this maneuver often elicits the "falling of the womb" sensation.

Of the many fruitless abdominal operations done because of symptoms due to ureteral stricture, that for supposed appendicitis heads the list. If one simply keeps in mind the usual location of the appendix near the anterior-superior iliac spine, and the location of the ureter near the umbilical region, this mistake will not occur so frequently in the future. The ureter, at the pelvic brim crossing, lies over the anterior portion of the sacro-iliac joint and one may have difficulty in differentiating by palpation a tender ureter from an arthritis, but we gain additional knowledge by palpation of the joint posteriorly and especially by palpation of the ureter in its juxtavesical portion.

In attempting to differentiate an obscure abdominal complaint by the aid of palpation one must not lose sight of the fact that ureteritis is of such common incidence that it frequently occurs in association with the various other abdominal and pelvic diseases; and it is the duty of the diagnostician to evaluate the various obscure signs and symptoms and to advise the patient as to the sequence of the proposed lines of treatment.

I have briefly outlined a few of the outstanding features you are to bear in mind in your attempt to place a helpful diagnostic label on some of your patients heretofore directed to the wrong specialist or nursed under your own care as neurasthenics. In patients complaining of obscure symptoms referable to a possible lesion in the back, abdomen, or pelvis, you are to keep an open ear to any complaint of bladder trouble, an open eye for any suggestion of pathological findings in the urine, and inquisitive finger tips for any signs of soreness or tenderness along the urinary tract.

Time forbids a full discussion of the many interesting points in

differential diagnosis. There is probably no other local disease which gives rise to such protean symptoms as does ureteral stricture. Many of its more general manifestations result, of course, from its effects on the upper urinary tract. Because of its usual location in the pelvis its local discomforts are most often charged to the internal genitalia. From the involvement of the neighboring nerves we have referred symptoms upward to the flanks, posteriorly to the sacral and sciatic regions, laterally to the hips, and downward to the groin, thighs, legs, and even to the toes. The downward radiations within the pelvis give rise to annoying bladder, uterine and rectal symptoms, and the pains referred to the vagina and perineal regions often lead to misdirected operations.

As a result of the urinary stasis we have all grades of damage to the kidneys, with the resultant local and referred renal pains, and widespread general symptoms such as headache, fever, malaise, thoracic pain, and gastrointestinal symptoms.

The internists who have given attention to this disease report to me their amazement at the number of chronic gastrointestinal complaints which are traceable to defective urinary drainage, and at the satisfactory results obtained from treatment. I cannot emphasize this point better than by quoting from a table in a previous publication on "End-results in 100 Cases of Ureteral Stricture."[1]

Complaint.	Number of patients.	Relief, partial or total.	Per cent.
Indigestion	24	17	71
Anorexia	26	20	77
Nausea	56	25	45
Gas	42	26	62
Diarrhea	15	9	60
Rectal pain or pressure	28	19	68
Mucous colitis	15	12	80

Our title calls for a discussion of the pathology and treatment of ureteral stricture, but our time is limited. Those interested in the pathology are referred to a recent publication by Hunner and Wharton—"The Pathological Findings in Cases Clinically Diagnosed as Ureteral Stricture"[2] also to the publication by Goldstein and Carson—"A Study of the Urinary Tract in Autopsy Specimens, Correlation of Anatomy, Pathology and Roentgenology."[3] The chief interest from the pathology viewpoint lies not in a study of the simple inflammatory stricture area of the ureteral wall, but in a study

[1] Jour. of Urol., 1924, xii, 305. [2] Ibid., 1926, xv, 57. [3] Ibid., 1926, xv, 155.

of the many renal lesions resulting from the stasis of urine caused by the ureteral disease. The more common renal diseases which in large measure may be traced back to the injury caused by ureteral stricture are hydronephrosis (sterile or infected), chronic pyelitis and pyonephrosis, pyelitis of the pregnant and puerperal periods, chronic pyelitis in children, renal and ureteral calculi, the renal inflammatory processes resulting in the so-called essential hematurias and the various pathological processes which drive to the urologist the patient with congenital malformation of the upper tract. It is probable that many of the chronic nephritides resulting in multiple abortions are secondary to stricture as are some of the chronic renal processes formerly considered as amenable only to medical methods of treatment.

Inasmuch as the renal injury in the above list of pathological processes arises largely from defective drainage it is a mere truism to state that the first requirement of intelligent treatment is *the establishment of good drainage*. Focal infections are not only responsible for most ureteral strictures, but they sometimes contribute factors of direct injury to the renal substance, thereby furnishing two urgent reasons for the early discovery and eradication of possible foci of infection as one of the primary requisites of treatment in dealing with many of the above renal diseases.

Conclusions.—Ureteral stricture is one of the most common lesions of the abdominopelvic cavity. The symptoms due to its presence are most protean in character and lead to more errors in diagnosis and to more ill-directed therapeutics and unnecessary operations than those of any other disease.

The attendant anamnesis and physical findings are such that the general practitioner, if he but have the disease in mind, can usually make the diagnosis with even more certainty than obtains in dealing with cases of chronic appendicitis or chronic cholecystitis.

The chief interest to the pathologist centers in a study of the renal lesions caused by the presence of ureteral stricture.

The treatment is usually nonsurgical and consists in relief of the local symptoms and improvement in the kidney function, by dilatation of the stricture area and restoration of adequate renal drainage. Clinical experience seems to point to distant foci of infection as the most frequent cause of ureteral stricture, and treatment often fails to give permanent relief until the original focus of infection is eradicated.

DISCUSSION

Dr. Brooke M. Anspach: Dr. Hunner's papers usually give evidence of careful study and preparation and this one has been no exception. I have heard Dr. Hunner on this subject before. I think I heard him when he presented his first paper, and among those present there were many who expressed great skepticism over the existence of ureteral strictures, or ureteral narrowings, which give rise to symptoms. It occurred to me at that time that possibly the difference in opinion might be due in some measure to the methods used in examining the ureter, because I knew that Dr. Hunner used the Kelly cystoscope, which is the direct method of exposing the ureter, the patient being in the knee-chest or Sims' position, and the holding of the catheter or bougie directly in the hand, so that the difficulty in introducing or withdrawing the catheter is much more easily perceived. I do not know whether the direct method is possible in the male, but it has this distinct advantage over the indirect method in the female.

It has been my good fortune to be associated at the Jefferson Hospital with Dr. H. M. Ginsberg, who uses the direct method of cystoscopy; we have made a serious effort to study the problem which has been propounded by Dr. Hunner and see how often what he calls ureteral strictures, or ureteral narrowings, may be responsible for symptoms that might be mistaken for evidence of other and more common lesions. As a result of our study, I have no doubt at all that alterations in the caliber and functional activity of the ureters do occur much more often than we imagined before Dr. Hunner's original work. The plan we have pursued in our observation is about as follows: If a careful history reveals symptoms that may be ascribed to the urinary tract and are not easily explainable on other grounds, and especially if at the same time a specimen of urine taken from the bladder by a catheter shows an appreciable number of pus cells, even as few as three to five pus cells to a high-power field, we make an examination of the ureter on the affected side or on both sides.

As a result of this study of our cases clinically, and after examining the ureters at the postmortem table, we feel that if there is a "hang" when a catheter armed with a 3¼ mm. wax bulb is withdrawn from the ureter and the patient complains of pain similar to the obscure pain under investigation, and if there is pus in the urine, a tentative diagnosis of ureteral stricture, or ureteral narrowing, whichever you choose to call it, is justified, and we investigate the case further with ureterograms and pyelograms. I think it is quite true that unless one is constantly on guard, he will overlook these affections of the ureter which may be responsible for pain and operations will be done which prove to be useless and which were in the beginning unnecessary.

I have here a list of cases which have come under observation in which the original complaint of the patient was due to an undiscovered ureteral disorder and continued unrelieved by operation and other plans of treatment until the ureteral trouble was detected and treated. I will not detail these cases to you, as the hour is late. Suffice it to say that there were some in

which such organs as the appendix, the gall-bladder or the ovary were removed, or the uterus was suspended, or some other operative procedures were carried out but without relief until examination of the ureters had showed a stricture or a narrowing, and dilatation of the ureter had restored its caliber. An interesting case, suggesting ureteral narrowing as the result of focal infection, which has recently come under my observation was that of a woman who complained of pain in the right lower abdomen. She had had one rather difficult labor; there was a tear of the cervix, a relaxation of the perineum and descensus of the uterus; the adnexa were normal, and the pain could not be entirely explained by the pelvic condition. A catheterized specimen of urine showed pus, and on examination there was a very distinct narrowing of the lower part of the right ureter. The ureteral specimen showed pus and an infection with the staphylococcus. Curiously enough, this woman had had a paronychia of the first and second finger of her left hand for two years, which recurred periodically, and a culture of the pus from the finger-nail a year previously had shown the staphylococcus.

Now I think there is great danger in being overenthusiastic about ureteral narrowing or stricture and in ascribing to it certain symptoms; we must not forget this danger; we should pursue a sane and middle course. I wish to repeat that I would attribute symptoms to the ureters only when the examination shows what I have previously described, and I would be loath to attach major importance to the ureteral condition if some other more common and well-marked cause of the suffering were evidently in existence. In one of our earlier cases we thought we found a ureteral narrowing which explained pain on the right side, but this woman did not show evidence of inflammation along the ureters, having no bacteria or pus in the urine, and in making an examination under anesthesia we found an ovarian cyst with a partially twisted pedicle. After removal of the cyst the patient had no more pain. In making a ureteral examination of this case the patient had declared that the pain produced by it was exactly like the pain of which she had complained. Such an experience shows that one dare not draw hasty conclusions, and that a diagnosis of ureteral stricture or ureteral narrowing is only justified after the most thorough and painstaking exclusion of other causes.

While I might not agree with Dr. Hunner in his interpretation of the individual case, nevertheless, I think he deserves great credit for persistently bringing this subject before the profession and that he has rendered a very valuable service.

Dr. Alexander Randall: It is always intensely interesting to hear a man present a subject on which he is an enthusiast and on which he presents an authoritative opinion. It has been delightful to see Dr. Hunner throw himself into the subject of ureteral stricture and to travel with him over its symptomotology and diagnosis, as likewise to hear him present the interesting case reports and results of his therapy.

I find myself in a peculiar position, as being one thoroughly interested in the subject and yet one who has seen a comparatively small number of

cases of ureteral stricture, at least I have been able to satisfy myself with a diagnosis of such a condition in only a few cases. Frankly, I have not followed Dr. Hunner's methods of diagnosis, and possibly that is the reason, as much as anything, why I have not found the condition to be as frequent as he has.

The subject of ureteral stricture has been up for discussion a great many times since Dr. Hunner first drew our attention to its existence ten years ago, and after this period of time there still exists a large group of urologists and gynecologists who do not accept the condition to be as frequent in its occurrence, nor to present the symptomatology to which Dr. Hunner ascribes it, and I find myself tonight more or less forced into the position of the Devil's Advocate in attempting to discuss his presentation. I do not know, Mr. President, whether or not you are familiar with the duties of a Devil's Advocate, but before a person can be canonized, such canonization is subjected to a searching inquiry. If such inquiry proves the individual worthy of this distinction, there is then appointed an "advocatus diaboli," whose disagreeable duty it is to likewise search the individual's record in an effort to find any reason why such canonization should not be allowed. So, if I may, let me bring forward the ideas of those who do not agree with Dr. Hunner, so that this audience may be presented with the facts from both points of view.

As I have stated above, Dr. Hunner first presented his subject ten years ago, and, as far as I know, it was not until last spring, at the meeting of the Triennial Congress at Washington, that his first convert presented findings which ran closely to those of Dr. Hunner's. This was Dr. Rathbun, of Brooklyn. The discussion of Dr. Rathbun's paper was interesting. It was opened by Dr. Livermore, of Memphis, who likewise had been getting results similar to Dr. Hunner and was likewise enthusiastic over the diagnosis and the frequency of the occurrence of ureteral stricture. Dr. Hunner next took part in the discussion, and it seemed as though a wave of enthusiasm was starting in favor of the frequent occurrence of ureteral stricture. As the discussion progressed, Dr. Braasch, of Rochester, brought forward the idea that a number of these cases were due to ureteral spasm, which would give the same hang with a large wax bulb which Dr. Hunner uses and be interpreted as stricture. Dr. Young, of Baltimore, suggested their occurrence in the male as secondary to an underlying seminal vesiculitis. Dr. Crowell, of North Carolina, felt that one ureterogram should never be taken as conclusive, but that the contracture should be found on repeated examinations at exactly the same spot. Dr. Lower, of Cleveland, likewise would not accept its occurrence unless repeated ureterograms showed identically the same readings. Dr. Thomas, of Minneapolis, had examined 150 bodies at autopsy, searching for ureteral stricture, and had found but one present. Dr. Graves, of Boston, spoke of the systole and diastole of ureteral peristaltic contraction and the ease with which such could be misinterpreted. And so the discussion went, showing a large group of representative men who were adverse to accepting a frequent occurrence of these apparent strictures, especially when diagnosed by the methods advocated.

First, let me state the conditions which produce stricture and which all recognize: (1) Strictures of congenital origin; (2) those due to tuberculosis; (3) those secondary to calculus; (4) those the after result of acute trauma. These strictures everyone is willing to accept.

I wish to deal tonight only with that group which Dr. Hunner calls "simple chronic ureteral stricture," and wish not to be so drastic as to say that such do not exist at all, but to take exception to the frequency of their occurrence and to his statement that "it is the most common of intraabdominal lesions." First, let me point out that Dr. Hunner finds these (in his own statistics) where in 66 per cent of the cases there was likewise found sterile urine, or perhaps better stated, that in only 33 per cent was the urine found infected, and that in 46 per cent of his cases its occurrence was in the nulliparous.

As regards the terminology of what he means by ureteral stricture: Though we see stricture in other hollow organs, the only near corollary is that of urethral stricture, and I dare say that nine out of every ten men hearing the word used call to mind the more familiar picture of urethral stricture. Is there any similarity? The urethra is a urinary passage having small and practically nonfunctioning musculature, is lined by a mucous membrane studded with lymphoid tissue and numerous small mucous glands. It lies in an organ subject to any amount of external trauma, suffering innumerable insults, functioning as a urinary passage about 1 per cent of the time and the canal itself the frequent seat of a characteristic acute infectious disease, and it is after this acute infectious disease that we see its characteristic strictured condition. This pathology is well known and is recognized to be the result of chronic infection. The ureter, on the other hand, though again a urinary passageway, is a simple tube with a well-musculatured wall, 99 per cent of the time constantly functioning by peristalsis, lined by a mucous membrane devoid of mucous glands or lymphoid tissue and marvelously well protected from trauma and rarely if ever the primary seat of any infectious process. So in this terminology one misses any anology to strictured conditions seen elsewhere and brought to mind by the use of this term.

However, accepting the terminology as implied when speaking of ureteral strictures, let us turn to the pathology of ureteral stricture as it is known today. Perfectly frankly, it does not exist. Dr. Hunner was rushed tonight in his presentation and referred but briefly to a recent article in last month's issue of the *Journal of Urology*, in which he has presented this phase of the subject under the title of "Pathological Findings in Cases Clinically Diagnosed as Ureteral Stricture." I imagine that Dr. Hunner himself is not satisfied with this study, for it presents the autopsy findings of but 8 cases. In 3 the stricture is classified as being of congenital origin. This is one of the types granted. In 3 more patients the condition was found in association with an extensive malignancy of the pelvic organs. These again are not the simplest ureteral strictures with which we are dealing. In the seventh case, put down as one of infectious origin, the patient died of extensive urosepsis with advanced cystitis, ureteritis and pyonephrosis; while in the eighth case, again classified as of infectious origin, the specimen

was lost to study and it is mentioned that the condition found in the kidney suggested some unusual form of parasitic disease. The article proves nothing. To anyone in this room tonight it is open to prove this simple problem, for a few months spent attending the routine autopsies at any large municipal hospital should give the answer. This has not been done, and I believe that I might summarize the feelings of all urologists that until this important corner-stone in the study of any diseased condition has been firmly and scientifically laid, no one will be willing to accept a superstructure erected thereon, for truly all clinical medicine and clinical surgery must rise upon a foundation of gross and microscopic morbid anatomy, if it is to stand the test of time and gain the acceptance of the scientific world.

Dr. Huxner (closing): I wish to thank the gentlemen most sincerely for their discussion. I think that if one has any truth in his contentions he does not need to worry much about discussions. We have had two types of discussion, that by Dr. Anspach, who with Dr. Ginsberg has been investigating this subject and in the broader principles agrees with my conclusions; and second, that of Dr. Randall, who represents the position of most of the prominent urologists of the country who have refused to investigate, and who are still satisfied with swivel-chair arguments against the conception.

Dr. Randall has stated quite fairly the unsatisfactory character of the pathological material recently presented as bearing on this subject. Dr. Wharton and I expressed our dissatisfaction in putting out this material, which in 8 cases represented only 2 belonging to the focal-infection type of stricture. We spoke of the difficulties of getting these specimens at operation, and if properly treated the patient does not often come to autopsy. Even in some of our cases that came to autopsy with a clinical diagnosis of stricture the pathologists overlooked the essential lesion and registered the diagnosis under congenital hydronephrosis, or in line with some of the traditional views.

I have felt confident that stricture must be found at autopsy if looked for, because I have seen and handled many strictures at operation, and have had my assistants at the operation palpate these areas of chronic infiltration in the ureteral walls. In some cases where the wax bulb has located an area of infiltration in the ureteral wall, and I have had opportunity later at operation to look for stricture, there has not been sufficient infiltration to make a palpable mass under the gloved-finger palpation. I do not interpret this, as do my urological friends, as a case of ureteral spasm, but as a case in which the bulb has detected an area of infiltration so slight that it cannot be palpated. We occasionally have the withdrawing catheter gripped by a spasm of the trigonum, but if we get a definite scar-tissue grating sensation as the bulb comes through a certain area in the ureter, and this "hang" is always repeated in the same area in the individual ureter, we can scarcely call this ureteral spasm.

I am thankful to Dr. Randall for his frank discussion, for I am sure that is what we all wish on any new issue. If he will examine next month's issue of the *Journal of Urology* for the article of Goldstein and Carson on

their autopsy findings in the past three years, I am sure he will find some surprising results. I have been confident that strictures would be found at autopsy if proper means were used for their discovery when present, but I would not have dared to predict that 9 per cent of autopsies examined in this careful manner would reveal definite scar-tissue stricture, verified by microscopic examination, and that of 22 out of 90 ureters showing gross changes by visual and urogram examination, all of them would show pathologic changes under the microscope.

Thus far, ureteral stricture work has been undertaken largely by surgeons and gynecologists over the country who are doing their own urology, and who have been glad to find another method of perfecting their diagnosis in obscure abdominal and pelvic conditions and thus avoid fruitless operations. I am glad to assure this audience of internists that the urologists are coming around one by one.

EXPERIENCE WITH MALARIAL TREATMENT OF SYPHILIS*

By JAY F. SCHAMBERG, M.D.

AND

SIGMUND S. GREENBAUM, M.D. (By Invitation).

The malarial treatment of paresis was introduced by Wagner-Jauregg, of Vienna, in 1917, after many experimental trials with other fever-inducing inoculations. He has treated in his clinic over 1000 cases of paresis with malarial inoculation.

Of the first 9 cases treated in 1917, 4 developed remissions and 3 were back at their vocations for six and a half to seven and a half years, up to the time of the last publication.

In the group treated in 1919–1920, 25 cases, 18 had complete remissions with ability to return to work; 5 had remissions lasting for five years (the time of last publication) and 2 more were working. In 1920–1921 there were 116 cases, of whom 78, or 67 per cent, had remissions: 38 of the cases, or 32.7 per cent, were well for a period of four to four and a half years, up to 1925.

In 1921–1922 there were 250 cases of paresis treated, with 166, or 66 per cent, remissions; 83, or 33 per cent were complete remissions.

Nonné, of Hamburg, in the last edition of his well-known book, *Syphilis und Nerven-system*, gives a report of 96 clinically and serologically proven cases of paresis treated with malarial inoculation: 48 per cent were enabled to return to work; later this number was increased to 68 per cent.

In England and the United States the percentage of remissions has been quite comparable with the original results obtained in Vienna.

Personal Results. The results herein reported have been obtained on patients observed over a period of eighteen months. European observers of the effects of malarial therapy in neuro-

* Read March 3, 1926.

syphilis, and more particularly in dementia paralytica, stress the importance of the time factor subsequent to the treatment. Many of the treated cases fail to show either clinical or serologic improvement for many months. Those using this type of therapy should keep this important point in mind because of its bearing on the prognosis. However, a small proportion of the patients show definite improvement either during the last malarial paroxysms or shortly after their cessation.

Our studies were made upon patients with paresis, tabes dorsalis, cerebrospinal syphilis, both diffuse and localized, and congenital syphilis. The total number of patients in the study is 53. We have treated more than this number, but there have been excluded from this report patients who passed through abortive attacks of malaria or who have been so recently treated as not to warrant their inclusion.

Results in Paresis. All of these patients, as well as the others with various types of neurosyphilis, were examined and the diagnosis confirmed by competent neurologists.[1] In all of the paretics the serologic and the neurologic changes were more or less uniform. The mental symptoms, however, varied from a simple euphoria to the severest maniacal and depressive manifestations. Estimation of the value of fever therapy in these paretics has been based not only upon the neurologic and laboratory findings subsequent to the treatment but more particularly upon the mental results. For various reasons the term cure is best avoided; the preferable terms are complete remission, partial remission (temporary or followed by a very gradual but definite improvement ending in either a complete remission or as an arrested case with definite mental changes still present), and last, patients in whom the treatment had no effects whatever. In this connection it should be emphasized that this treatment, in not a single instance, had deleterious effects on the paresis itself.

In our group of 14 paretics, representing all the types of the disease, there was a complete remission in 4 patients and partial remission in 6 patients (1 of which was temporary); 2 of the latter are still improving and the other 3 stationary; 3 were uninfluenced by the treatment and 1 died. The death occurred very early in our series and took place in an advanced paretic who developed trophic

[1] Drs. Potts, Patten and Yaskin.

lesions on the pressure points of the shoulders, buttocks and heels, and subsequently a bronchopneumonia.

Generally speaking, mental alterations in paretics are of several classifiable types: These are the maniacal, expansive, depressed with progressive deterioration and the paranoic. In our experience, and in the experience of others, it is the first two types which are specially responsive to malarial therapy, despite the fact, as has been pointed out, that these types show very slight tendency to spontaneous remissions. Of the 4 patients with complete remissions, 2 were of the expansive and 2 were of the maniacal type.

The patients classed as complete remissions are, at the present time, to all intents and purposes, mentally normal and are all back at work. In one, return to mental normality occurred shortly after the cure of his malaria and he has remained normal for over fifteen months. This patient was a man, aged thirty-five years, with classic serologic and clinical evidences of paresis. He had delusions of grandeur and was so violent during his hospital sojourn that he had to be restrained hand and foot. This patient returned to his home perfectly rational and has resumed his work as a civil engineer, giving entire satisfaction for the past sixteen months. His blood Wassermann has become negative and the colloidal-gold test in the spinal fluid, which was characteristically paretic, has changed to a luetic type. The spinal Wassermann is still positive, but not as strong as it was originally.

The physical improvement in this group, and in more than half of those who showed partial remission, was commensurate with the psychic amelioration in the direction of speech defects, sphincter control, libido and muscle tremors. Reflexes and pupillary changes remained uninfluenced or were but slightly altered. In one of those with complete remission, however, an Argyll-Robertson pupil disappeared and a hyperreflexia became normal. One of the most striking improvements in many of the malarial-treated patients, in all forms of neurosyphilis, is the wonderful feeling of well-being and the progressive gain in weight subsequent to the treatment. Several of the paretics weigh from 15 to 20 pounds more than their best previous weight. This gain in weight is not always associated with mental improvement. In one paretic, classed as a partial remission, with very marked somatic complaints, filthy habits, memory disturbances, all is normal except for a single complaint regarding inability to digest his food. (Chart I and II.)

Chart I.—Spontaneous Remissions in Paresis (*From Gerstmann*)

	Per cent.
Acker (1888)	14.30
Behr (1900)	4.08
Jarhmarker (1901)	14.80*
Mattey (1901)	7.10
Hoppe (1901)	15.00
Gaupp (1903)	10.00
Kraepelin	15.90
Joachim (1912)	4.00
Dubel (1916)	13.50†
	6.50*
Weichbrodt (1920	10.00
Weggendorfer (1921)	13.00
Kirschbaum (1923)	11.70
Sophoff (1924)	4.80

Average, 10.6 per cent. * Women. † Men.

Chart II.—Clinical Results of Malarial Therapy in Paresis

Wagner-Jauregg: Treated 9 cases of paresis in 1917. There resulted 4 complete remissions; 2 incomplete remissions; 2 uninfluenced remission; 1 died during febrile period. Three of these patients at work at the present time, six and a half to seven years later. Thirty-three per cent.

Wagner-Jauregg: Treated 25 cases of paresis in 1919–1920. There resulted 18 remissions: 7 were complete; six were incomplete; 5 were of mild grade. Of these, 8 are at work—five years later. Thirty-two per cent.

Wagner-Jauregg: Treated 116 cases of paresis in 1920–1921. There resulted 78 remissions, of which 42 were complete. Thirty-three per cent at work and in complete remission—four to four and a half years later.

Nonné: 44 per cent in complete remission and at work.

Personal Results: 28 per cent in complete remission.

We were able to make subsequent serologic studies in from four to twelve months in all of those with complete remission and in some of those with partial remission. In agreement with other observers, the serologic alterations for the better bore no constant relation to the mental changes. In one of those classed as a partial remission, both spinal fluid and blood became entirely normal within four months after the treatment although the mental condition remained uninfluenced. Eight months later, the serologic conditions remain negative, while the mental status is stationary and does not as yet show any of the progressive deterioration his type usually undergoes.

In those in whom complete remissions occurred, favorable serologic changes were found usually in the cell count, globulin content and colloidal-gold reaction with the spinal fluid complement-fixation reaction being as a rule less influenced. The cell count was the first and most commonly observed change, dropping, as a rule, to very nearly normal in fluids originally showing from fifty to several hundred cells. (Chart III.)

CHART III.—SPINAL FLUID CHANGES SUBSEQUENT TO MALARIA INOCULATA.

Number.	Disease.	Before.				Time interval, mos.	After.				Mental result.
		Cells.	Globulin.	Gold sol.	Wassermann.		Cells	Globulin.	Gold sol	Wassermann.	
1	Paresis	–	–	5555555421	4+	14	–	–	0112312100	3+	Complete remission
2	"	33	–	4411000000	4+	5	4	–	1111000000	4+	Complete remission
3	"	42	–	5554424200	4+	4	22	–	5555432200	4+	Temporary remission
4	"	30	+	0	4+	8	14	–	00	Negative	Partial remission
5	"	81	++	1111221000	4+	6	13	–	1112000000	4+	Complete remission
6	"	184	+	5544322221	2+	5	14	–	3322110000	4+	Marked partial remission
7	"	2	–	5554422111	3+	8	6	–	2211000000	2+	Complete remission
8	"	24	+	3322211000	4+	4	5	–	455543210	Negative	Temporary remission
9	"	–	–	5543455553	4+	10	–	–	5555554210	4+	Uninfluenced
10	Tabes dorsalis	21	+	–	4+	6	3	–		3+	
11	" "	47		–	3+	5	6	–		2+	
12	" "	0	+	–	4+	3	3	–		Negative	
13	" "	0	+	–	3+	8	7	–		Negative	
14	Congenital syphilis	–	–	344332211	4+	6	–	–	2111100000	Negative	
15	Cerebrospinal syphilis	21	+	3333330000	4+	6	10	–	1122110000	3+	
16	" "	23	+	2554331100	4+	5	11	–	2111110000	4+	
17	" "	88	–	–	4+	6	5	–	–	2+	
18	" "	47	–	–	3+	5	6	–		2+	
19	" "	–	+	–	2+	7	–	–		Negative	
20	" "	147	+	–	4+	5	–	–		Weak +	
21	" "	158	+	1123211000	4+	4	6	–	0	Weak +	
22	" "	117	++	1111122100	3+	5	41	+	5554321000	2+	
23	" "	21	+	–	4+	9	11	–		Negative	
24	" "	65	–	10000000011	4+	6	7	–	3332100000	4+	

2. *Results in Tabes Dorsalis.* These patients were treated with two objects in view, namely, to control symptoms resistant to the usual antisyphilitic remedies and to arrest the progress of the disease.

Seven tabetics out of ten treated went through a course of malarial paroxysms considered therapeutically sufficient. Of these 7, 4 had active subjective symptoms, consisting of gastric crises in 3 and shooting pains in the extremities in 1. In all, the pains became decidedly worse and almost unbearable during each malarial paroxysm. When discharged, however, all felt better, and stated that the result justified the suffering through which they had gone. They were practically free from pain. One with gastric crises, could not be traced subsequently; but the remaining 2 with gastric crises have been symptom-free for six months and two months respectively. The one who had been symptom-free for six months had had two minor attacks during this time, but they disappeared spontaneously in several days. Neither of these patients had ever had more than three or four weeks of freedom from pain since the onset of their condition. The ataxia in this last patient, and in another with severe pains in the extremities, has shown definite improvement. The latter patient has been pain-free for two months, and according to his own statement feels well repaid for going through the "cure."

In two tabetic patients there had already developed a partial optic atrophy. In neither instance was this influenced except temporarily and subjectively. This has progressed, as it so commonly does, to complete blindness.

Serologic studies were made from three to six months after the malarial course. In those in whom such studies were possible, improvement in the pleocytosis and globulin content were noted, and in one instance a spinal fluid which has been strongly positive before treatment and had shown a definite increase in globulin was found to be completely negative three months later (Chart I).

3. *Results in Congenital Syphilis.* It is generally recognized that congenital syphilitics often fail to respond to the usual antisyphilitic remedies in the sense that seropositive patients often remain so despite the most efficient and prolonged treatment. This is particularly true in those who have never had antisyphilitic treatment before and in whom the disease is old. In general it may be said that the older the syphilis, as in the acquired form, the more difficult it is to obtain negative Wassermanns.

We selected for this study cases such as the above, that is, untreated old congenital syphilities, as well as those deemed "Wassermann-fast" after prolonged treatment with the usual drugs.

Only 7 of those treated developed a sufficient number of malarial paroxysms to warrant inclusion in this study; 5 of these were chronic congenital syphilities who had had little or no treatment and 2 were considered "Wassermann-fast." The longest period of observation since malarial inoculation has been one year. In neither the "Wassermann-fast" nor the chronic untreated syphilitic was the complement-fixation or the precipitation reactions influenced.

In two, diagnosed juvenile tabes, definite spinal-fluid changes were present before the treatment; one of these patients had so marked an ataxia before treatment that he was practically bedridden; seen six months later, his ataxia was barely noticeable. The serologic results will be found in Chart I. Tertiary ulcerations of the leg of more than a year's duration in one of these patients healed completely by the tenth malarial paroxysm.

4. *Results in Cerebrospinal Syphilis.* This group consisted of 25 patients. The objects sought were the determination of the effects upon acute lesions or the palsies, the arrest of progressive second- and eighth-nerve atrophy, the effect upon the serologic abnormalities in the spinal fluid and, last, the prevention of paresis. The number of syphilities who will develop paresis is, according to most authors, less than 5 per cent. No one can, of course, foretell, which neurosyphilitic will become a paretic. However, it would appear to be safe to assume that, neurosyphilities whose spinal fluids are uninfluenced by intensive and prolonged treatment by approved methods, are potential candidates for paresis. We feel that such types should be given the possible benefit of this method of treatment.

Of the 7 patients who had optic atrophy, in various stages, all showed a temporary improvement, which was followed by progressive blindness. Those with acute palsies presented rather variant results. Two patients with third nerve palsies and one with bilateral seventh-nerve palsy were not influenced, whereas a fourth patient with paralysis of the left leg regained almost complete control of it by the ninth malarial paroxysm. A severe tertiary ulceration on the leg of one of them healed completely by the end of the malarial course. In two patients, one with a localized form of cerebrospinal

syphilis, causing constant nodding of the head, and the other, epileptic attacks, complete relief took place shortly after the cure of malaria. Both of these patients have been symptom-free for eight and ten months respectively. The patient with syphilitic epilepsy had been resistant to the usual antisyphilitic remedies for many months. The epilepsy has recently relapsed.

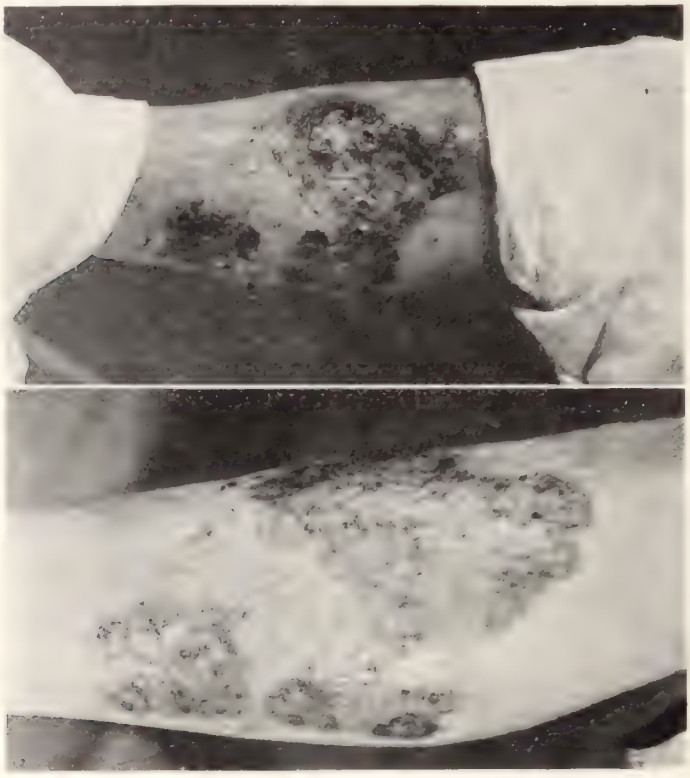

FIG. 1.—Tertiary ulceration on leg in patient with cerebrospinal syphilis. The lower photograph shows healed lesion one week after the last malarial paroxysm.

The serologic studies in these cases showed changes similar to those occurring in other forms of neurosyphilis after malarial therapy (Chart I). Several patients showed no spinal-fluid changes primarily, a few were subsequently lost track of, and a number refused lumbar puncture a second time.

CLINICAL COURSE AND TREATMENT OF MALARIA INOCULATA. Tertian malaria, the type used in this therapy, does not run a uni-

form course in each inoculated individual. Patients react differently to the infection irrespective of the clinical course in the donor. The fever may be a single tertian in the one and a double tertian in the next; in some instances two complete cycles may occur within thirty-six hours. Although the single tertian fever is more desirable than the quotidian type, in that it enables the patient to recuperate both physically and mentally for the next paroxysm, there is no means of constantly inducing this. In fact, the variability of the strain or of the immunologic reactions of the patient, or both, sometimes leads to abortion of the disease after two or three paroyxsms. In some instances there is no "take" at all. The negro is particularly resistant to malaria inoculata, but this resistance becomes less marked the lighter or closer to the Caucasian the particular negro is. The height of the temperature is likewise variable. In some it may not reach 102° F. and in others it may reach 106° F. or more. The temperatures, likewise, vary with each paroxysm, both in height and duration. The subjective symptoms are chiefly headache, vomiting and muscular pains. These symptoms vary in degree, the time of their onset in each patient and with each patient. Headache is usual, but vomiting is less common. Chills may be entirely absent, thus differing from natural malaria. In such cases the first sign of a malarial paroxysm is simply a febrile rise to 103° F. or more. As a rule, when chills are absent, it is only during the first few paroxysms. The highly neurotic patient suffers severely, and it may, at such times, be necessary to abruptly terminate the malaria or to ameliorate it with small doses of quinine. Paretics in our experience do not react this way. In fact, when rational, they usually express delight with the treatment. The loss of weight is variable and is naturally greater in those with severe vomiting and inability to eat as a result of the associated nausea. A successful malarial treatment should produce temperatures of 103° to 106° F., persisting at such points for from six to twelve hours.

As will be pointed out later, malaria inoculata is extremely susceptible to drugs. It is far easier to cure than is natural malaria. Thirty grains of quinine administered the first day and followed by 15 gr. a day for six succeeding days is usually sufficient. Neoarsphenamine may bring about the same result in doses of 0.9 gm., but reliance is preferably placed on quinine. The cure of the malaria is judged by the absence of the plasmodium from a blood smear prepared from blood concentrated in the centrifuge.

Inoculation, Incubation, and Number of Paroxysms. The infected blood is obtained from one suffering with natural or artificially induced malaria of the tertian type. Two or 3 cc. of blood, at any stage during the course of the malarial infection, is injected into the recipient either intravenously or subcutaneously. The intravenous route gives a much shorter incubation period. We have injected quite a number of patients, but have had a larger percentage of "nontakes" by this method. We have studied the corpuscle agglutinations in the donors and the recipients, and we are inclined to believe that some of the failures are due to cross agglutination. The subcutaneous method of inoculation is, therefore, preferable. The blood is injected in the back over the scapulæ or any other suitable place. This method will almost invariably give a "take," particularly if the strain of infecting plasmodium is at all active and it is the method of choice.

The incubation period varies from four to twenty-one days and averages about twelve days. At the end of this period there is, as a rule, a chill followed by a rise in temperature. This will occur daily or every other day. The number of paroxysms a patient is permitted to have varies from eight to sixteen, depending largely upon the physical stamina, age and complications.

Under certain conditions, reinoculation is necessary. This may be attempted in those whose paroxysms were insufficient, or in those in whom some improvement has occurred from the previous course and an effort is made to make still further gains. Reinoculation, after a short period, is not always possible. We have been successful in but a single instance out of five attempts.

Caution against use of Drugs during Malaria Inoculata. Natural tertian malaria is, as a rule, a benign disease and easily influenced by quinine. Artificial tertian malaria is even more easily influenced by this drug. A single grain of quinine hydrochloride will often inhibit or interrupt artificial malarial paroxysms for from twenty-four to forty-eight hours (Chart IV). Not only is malaria inoculata susceptible to small doses of quinine, but it is likewise susceptible to a number of other drugs, notably the coal-tar products. Acetanilide, antipyrine, phenacetin, amidopyrine, acetyl-salicylic acid or aspirin and allonal, etc., if given during the incubation period or during the paroxysms, are apt to abort the disease. Once inoculated, a patient should not be given any of these drugs. It is possible that other drugs will do the same thing.

It is therefore advisable to give a patient as few drugs as possible, and if narcotics or analgesics are necessary to use chloral or opium.

Complications and Contraindications. The use of malarial inoculation for therapeutic purposes is not entirely free of danger. It is not advisable to carry out this treatment unless the patient is hospitalized and under constant supervision. This is especially true for advanced paretics, in whom complications are particularly prone to occur. The following may be considered the most frequent of the complications: progressive weakness, herpes labialis; edema of the feet, ankles, face and hands; anemia, which is also the basic factor in the edemas; jaundice; delirium; hyperpyrexia and myocardial weakness. Many of our patients gradually developed, noticeable about the seventh or eighth paroxysm, a yellowish cutaneous tint common to malaria patients and not due to icterus. With the exception of hyperpyrexia and myocardial weakness, none of these disturbances, as a rule, call for termination of the malaria. The hyperpyrexia can be easily controlled, as has already been noted, and must be in advanced paretics in particular.

In advanced paretics, trophic lesions may develop with extreme rapidity. Large bullæ at pressure-points may be noted.

Tabetics with subjective or sensory disturbances should be warned that their pains are apt to be intensified during the malarial paroxysms. The postmalarial anemia and anemic edemas may persist for several months before the iron preparations used will control them.

Deaths as a result of this form of therapy have been reported as reaching from 6 to 8 per cent. Our single death gives us a mortality-rate far below this figure. It is our belief that the published percentages can be greatly reduced by careful selection of cases and close surveillance of the malarial infected patients.

Persons with moderate or severe visceral disease, such as myocarditis or renal disease, as well as elderly persons and very advanced emaciated paretics, are not suitable candidates for this type of therapy. In these latter a marked weakness may develop so rapidly as to necessitate termination of the malaria.

Treatment of Syphilis after Malarial Therapy. There is a diversity of opinion concerning the use of active antisyphilitic treatment subsequent to malaria inoculata. We have observed two paretics made decidedly worse, despite definite signs of previous improvement, when given injections of iron cacodylate and

neoarsphenamine shortly after the cure of their malaria. On the other hand, we have observed a tertiary ulceration, which had healed completely during the fever, spring into activity within one month of the malarial cure.

In Vienna most of the malarial cases are not given subsequent antisyphilitic medication, as in their experience it did not prove of value. There are others who feel that postmalarial antisyphilitic medication is definitely indicated, but ought not to be given for some months after the malarial cure. In the case of the paretic, if treatment is to be given, it should not be given before three months.

Summary and Conclusions. Twenty-eight per cent of the paretics treated have become for the time being clinically normal. Tabetics with various sensory disturbances have been definitely benefited. Optic neuritis in both tabetics and cerebrospinal syphilitics was not altered. Definite beneficial results are shown on certain cutaneous and cerebrospinal syphilitic lesions as well as on the spinal fluid.

Nonspecific therapy in syphilis has been widely advocated in recent years.[1] Such agents have been shown to be of value in aiding the present-day treatment of chronic syphilis. Malaria inoculata possibly represents one type of this form of antisyphilitic therapy. Our results, as well as those of other observers, are such that there can be no doubt of its value in certain forms of neurosyphilis and particularly in paresis. It would appear, both from a clinical and anatomicopathological point of view, that the best results should be obtained in early cases. The recognition of the fact that clinically early cases usually means fairly well-marked pathologic changes, is so important that we feel that those chronic neurosyphilitics in whom prolonged intensive antisyphilitic therapy has failed to bring about a return of the spinal fluid to normal, should be given the benefit of malaria inoculata if only with prophylaxis against paresis in view.

It is certain, moreover, that this type of therapy offers more hope of results than the usual drugs, irrespective of the stage of the paresis. Although sufficient time has not elapsed to enable us to determine the permanent effects in these latter cases, the immediate effects warrant its use in a condition hitherto admitted by all incurable.

[1] Greenbaum, S. S., and Wright, C. S.: Effects and Value of Nonspecific Therapy in Syphilis, Arch. Dermat. and Syphilol., November, 1924, p. 551; December, 1925, xii, 858.

DISCUSSION

Dr. Schamberg (closing): The mortality-rate of inoculation malaria is largely a matter of selection of cases. The higher number of fatalities in some of the reported series of cases was doubtless due to the type of paresis treated. As has occurred abroad, there were cases included in the group treated which were not best fitted for this form of treatment. In Europe, in the beginning, all types of paresis were treated with malaria, but later, cases of relatively short duration were particularly chosen. One thing is essential; that is, hospitalization of the patient. No patient should be treated with malarial inoculation in his home, as conditions very suddenly arise which may require an urgent termination of the malaria; therefore, close and constant surveillance of the patient is essential. Again, no patient should be treated who has not been subjected to a most thorough physical examination, particularly with reference to the heart. It is necessary also to make blood counts from time to time to guard against the development of an extreme anemia: this is one of the guides as to the permissible duration of the infection. Generally speaking, the more paroxysms the patient is allowed to have, the better the therapeutic result. Our first case of paresis that did so remarkably well was permitted to pass through fifteen malarial paroxysms, whereas the average is not much more than eight.

As regards the period of incubation of malaria inoculata, I agree with Dr. Wilson that it varies between two and twenty days, but averages about nine to twelve days. In order to economize bed space in the hospital, we primarily employed intravenous inoculation. This leads to a shorter incubation period, and therefore a lesser sojourn in the hospital. We found, however, that the percentage of failures was higher than inoculation by the subcutaneous route. From certain studies on agglutination, we believe that this was largely due to cross-agglutination of the blood of the donor and donee. Furthermore, it was difficult to successfully reinoculate by any method persons who had resisted an intravenous injection of malarial blood.

I believe that no one is in a position at the present day to express a definitive opinion as to the worth of malaria inoculata in syphilis. The evaluation of the method is in the making. Nevertheless, it is the opinion of eminent and conservative men abroad that it has achieved results in neurosyphilis not previously accomplished by any known method. We appreciate the fact that new methods of treatment are commonly accorded extravagant praise. Making due allowance for this tendency, we nevertheless believe that his method of treatment is pregnant with great possibilities in the treatment of syphilis of the central nervous system.

SURGERY IN DIABETIC GANGRENE*

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(By Invitation)

The relation of diabetes to surgical lesions has been frequently discussed. This relationship has been intensively studied from time to time because of the increased risk associated with an operation upon a diabetic, due to coma, susceptibility to infections and the tendency for an existing gangrene to extend. Complications and sequelæ often delayed or prevented the desired result.

Until recently the knowledge of the problems relating to the surgery of diabetics has been in a rather chaotic state. Some of the earlier opinions, often contradictory, will be cited to indicate how much the experience of different surgeons must have differed. Sternberg has stated that any necessary operation may be performed upon a diabetic with safety. It might be asked with justice, if Sternberg's statement is true, why any operation could not be performed. Lépine and Kausch, on the other hand, have stated that every operation is contraindicated. DaCosta states that the increased sugar-content of the blood made it a favorite culture medium, and as a result wounds in diabetics frequently become infected. Handman refutes this statement and demonstrated that the growth of staphylococci was the same in diabetic and non-diabetic blood, and that the bactericidal power of the blood is not influenced by the addition of 1 per cent of sugar. Manges believed that the surgeon had too much fear of acidosis and thought that there was no danger, however intense the acetone reaction, when the diacetic and oxybutric acid tests were negative.

These divergent opinions are hard to reconcile. Probably the severity of the diabetes differed quite markedly and the opinions may have been based upon a small series in which either the severe or mild cases predominated.

* Read March 3, 1926.

The introduction of insulin has stimulated the study of diabetes, and has directed attention to the fact that the diabetic, when occasion for surgery arises, should be considered as a borderline case, and that the internist and surgeon should coöperate in the closest way.

Surgery in the diabetic may be divided into two groups. (1) Those lesions in which diabetes seems to play an essential etiological rôle, such as perforating ulcer of the foot and different forms of gangrene. (2) The lesions occurring in a diabetic patient in which the diabetes is an incident – gall-stones, appendicitis, abdominal tumors, fractures, etc.

Perforating Ulcer of the Foot. A peculiar perforating ulcer of the foot occurs occasionally in diabetes. It is not unlike the perforating ulcer occurring in tabes, but is not identical with it. This lesion was first described in 1866. Later Naunyn described seven cases. Some of the cases described by Naunyn were not the typical perforating ulcer, for they were not typically located, did not have calloused edges or the punched-out appearance which are typical of the perforating ulcer associated with nervous lesions.

Some of these ulcers are described as beginning as a vesicle with a cloudy serum, from which developed a gangrenous inflammation which remained relatively superficial and extended but little. They usually develop upon the foot, but have been observed upon the upper extremity about the fingers and palm. In many instances the destructive process has not extended to the bone, and the lesion has healed when the diet has been modified, pressure removed and careful dressings applied. Occasionally an ulcer has developed upon the other foot, the original ulcer having healed.

As previously stated, many of these ulcers which have been reported as perforating ulcers cannot be accepted as such, if the term is employed in its strict sense, for they are not crater-like, do not have calloused margins and are not surrounded by anesthetic skin. These are, strictly speaking, ulcers which follow a circumscribed, phlegmonous inflammation of the skin, which have not healed because the diabetes has not been controlled.

There does occur, however, in diabetes a typical perforating ulcer not associated with tabes. The ulcer is usually situated upon the sole of the foot, just back of the head of the fourth metatarsal bone, or upon the under surface of the heel. It has calloused margins, is deep and crater-like, although not infrequently the skin surround-

ing it is not anesthetic. This ulcer because of its typical location and other characteristics resembles that observed in tabes. It is not unlike in general appearance the ulcer which may develop about the heel after division of the sciatic nerve. The ulcer may develop slowly or relatively rapidly. Diseased capillaries have been found in the skin adjacent to the ulcer.

This lesion occurring in diabetes is probably dependent upon the same factors as the perforating ulcer occurring in tabes. It is apt to develop at a point exposed to pressure. Nerve lesions, peripheral neuritis, *e. g.*, are not uncommon in diabetes, although the skin surrounding such an ulcer is not always anesthetic.

In not a few instances the destructive process extends to the head of the underlying metatarsal bone, usually the fourth. The head of the bone may be sequestrated, although no marked inflammatory reaction may be noted at the time.

There are several factors which combine to render healing of these ulcers slow or impossible. The patient is generally unable to remain off the foot; but rest in bed alone will not cause such an ulcer to heal. The rigid, calloused margins prevent the walls of the ulcer from falling together. This is an important factor in delaying or preventing healing. An *x*-ray should be taken to exclude the possibility of sequestration of the head of the bone, for if this has not occurred the ulcer may be excised, care being used to remove the calloused margins, and the resulting wound sutured. Naturally, the head, if sequestrated, should be removed. The wound should then be but partially closed. There is but little danger if the diabetes has been controlled before this procedure is undertaken.

It is quite probable that this lesion, resembling so much but not necessarily identical with the perforating ulcer of tabes, has the same etiological basis.

Diabetic Gangrene. The question has been repeatedly asked and experiments undertaken to determine why gangrene occurs so frequently in patients with diabetes. The relation between the two was first emphasized by Marschall di Calvi in the middle of the nineteenth century. The question has been variously answered. That a hyperglycemia and the metabolic changes associated therewith are not the sole cause of the gangrene, as was originally claimed, can be easily demonstrated, for the severest forms of diabetes, running a rapid course and terminating fatally frequently are unassociated with gangrene unless an infection develops. The more

chronic cases with a marked hyperglycemia often are not complicated by gangrene. It is quite probable, however, that one is unjustified in regarding the gangrene occurring in diabetics as a senile form and to deny a causal relationship between the gangrene and the diabetes.

Changes in the arteries are common in diabetics. Grube found changes in the arteries in 66 out of 177 diabetics, von Noorden in 261 out of 650 (40 per cent). Corner found arterial changes in 32.41 per cent. Gangrene observed in diabetics occurs at an earlier age than senile gangrene. The average age at which the so-called diabetic gangrene is observed is 54.4 years, while the average age at which senile gangrene is seen is 66.2 years. The greatest number of cases of diabetic gangrene are observed in the sixth decade, while senile gangrene rarely develops before the sixty-fifth year.

Different types of gangrene occurring in diabetics undoubtedly have different causes, but the gangrene most commonly observed in diabetics occurs in patients with vascular changes; the percentage varying from 65 to 100 per cent. It has been suggested that mild degrees of glycosuria occurring in patients with a marked arteriosclerosis might be due to sclerotic changes in the pancreatic vessels. This suggestion has never been widely accepted.

An examination of the arteries of extremities amputated for gangrene occurring in diabetics shows definite changes. They feel thickened and when cut the vessel wall is found to be definitely thickened. Many of the changes are most pronounced in the intima which not infrequently becomes separated from the media when the vessel is placed in fixing fluids. The inner surface of the artery is uneven, and atheromatous patches such as occur in senile arteriosclerosis are found. The larger arteries popliteal and posterior tibial which long remain patent may present the same changes. The intimal changes associated with definite elastic tissue changes are apparently compensatory and are most marked about foci in the media where degenerative changes have occurred. The thickening of the intima may be so marked that the lumen of the vessel is considerably narrowed, but it is not completely occluded until thrombus formation occurs. The degenerative changes may involve the media throughout the circumference of the artery. The specific elements in the media disappear. Where the media is thickened the intima is apt to be thinned. Softened, necrotic foci in the media become invaded by scar tissue. These areas may become calcified and finally turn into true bone. Vasa vasorum

penetrate toward these areas, and these minute vessels are surrounded by round cells which may take an active part in the transformation of this softened tissue into scar tissue.

The process goes on for months and years until the lumen of the vessels becomes occluded and gangrene develops. Where does occlusion leading to gangrene occur? Heidenhain has stated that in the noninflammatory type of diabetic gangrene occlusion occurs in the popliteal artery. This generalization is probably not justified, for anatomically, at least, there are cases of gangrene in which no occlusion of the popliteal artery is observed when an amputation is performed. The rather constant location of the gangrene would, however, suggest the obliteration of a large vessel at a fairly definite level. In some cases gangrene is due undoubtedly to thrombosis of the capillaries and smallest vessels, and although the popliteal and posterior tibial arteries are still patent their lumina are reduced in size.

Although the process is chronic there is very little collateral circulation established in these cases. This is quite different from thromboangeitis obliterans, another chronic vascular lesion associated with the development of a very marked collateral circulation. The definite changes occurring early in the media in arteriosclerosis, undoubtedly involving the vasa vasorum, may account for the differences in collateral circulation in arteriosclerosis and thromboangeitis obliterans.

All these changes are quite like those occurring in senile arteriosclerosis. They are more marked at an earlier age in the diabetic, gangrene occurring earlier. A weakening of the circulation becomes most pronounced in the distal part of the extremity as a result of the narrowing of the lumen. The bloodflow is lessened and finally ceases. Thrombi form in the capillaries and smallest vessels. Gangrene develops.

Diabetes undoubtedly has some direct etiological relationship to this type of gangrene, but the arterial changes do not differ from those noted in senile arteriosclerosis. Some slight inflammatory lesion associated with an abrasion, the trimming of a corn, the removal of an ingrowing toe-nail or the scratching of an eczematous area may be the determining factor in the early development of gangrene. The inflammatory lesion favors the development of a thrombus or the extension of a thrombotic process already established to one of the larger vessels.

The indications as to the procedure to be followed in cases of diabetic gangrene have become more clearly defined as the blood chemistry has been more carefully studied. Infection, when present, is often an indication for early operation, because of the inability to control the hyperglycemia by insulin. In these cases coöperation between the internist and surgeon is imperative, for the question has to be decided frequently whether or not the operation should be performed, even with a marked hyperglycemia. Coma may threaten and the removal of the gangrenous and infected extremity may be indicated, even when there is a possibility of coma following the operation, in order that insulin may become more effective. The general condition of the patient must be taken into consideration in determining when the operation should be undertaken, for the blood chemistry, if relied upon entirely, would contraindicate such a procedure.

Infections increase the normal blood-sugar. They produce the same effect, but to a greater extent in diabetes. Patients not suffering with diabetes, when subject to chronic infections, may have marked changes in sugar metabolism, characterized by an increase in blood-sugar beyond the normal amounts after ingestion of glucose and a prolongation of the blood-sugar curve beyond the normal two hours. The tendency to acidosis is more marked. A diabetic patient may have progressed in a most satisfactory manner for a considerable period of time and may have acquired the ability to utilize a considerable amount of carbohydrates, the blood-sugar remaining for the time being practically normal; but if an infection occurs the blood-sugar increases and sugar may appear in the urine, even though the patient is scarcely able to take any food.

It is in these cases, in which an infection is superimposed upon a gangrene, that an operation may be indicated to remove the infection and thus permit of the control of the hyperglycemia. The indications for operation, which may be distinctly contraindicated by the blood chemistry, must be determined by the experience and judgment of the surgeon in charge.

The surgeon must, however, be in the possession of a few fundamental facts if he expects to meet the surgical indications arising in the diabetic wisely and correctly. While he may be able to arrive at correct conclusions and base his judgment upon gross clinical data, upon whether the hyperglycemia can be easily controlled by diet without disturbing too much the metabolic equilib-

rium of the patient, there are certain laboratory findings upon which in the end he will have to base his conclusions.

In the normal fasting individual the blood contains 0.1 per cent of sugar. This is not a fixed percentage. A perfectly normal individual may have as much as 0.1 or even higher percentage. The renal threshold for sugar is 0.17 to 0.18 per cent of blood-sugar. When the percentage of blood-sugar rises above the normal limit, sugar passes through the kidney and appears in the urine. The point of concentration at which the first trace escapes through the renal epithelium and appears in the urine is known as the kidney threshold. The process of escape is know as the renal leak. When a patient has a normal blood-sugar and sugar nevertheless appears in the urine, he should probably be regarded as a case of renal diabetes. For some reason or other his renal epithelium is permeable to sugar. It is questionable, however, whether it can be stated without reservation that such a patient is a better operative risk than one which is suffering with an essential diabetes.

The patient with an abnormally high blood-sugar, with no sugar in the urine, is the one that must receive the most serious consideration when surgery of the diabetic is discussed. This group unless recognized and carefully handled will probably contain the patients in which the surgical disaster occurs quite unexpectedly. They may give the history that they have had diabetes for which they have been successfully treated or from which they have recovered.

There may be no sugar in the urine and the patient seems to be a fair operative risk; but coma is apt to follow operative interference. Most of these patients have a mild, some an advanced nephritis. Nephritis raises the renal threshold. The blood-sugar may be abnormally high and still no sugar appears in the urine. There is no need to discuss the advisability of a blood-sugar determination in all suspected cases. It has been stated that a fatal result always follows major operations on patients whose blood-sugar is over 0.35 and whose blood CO_2 combining-power is less than 40. As previously stated, it may be necessary, even when the risk is high, to undertake an operation, for the blood-sugar may be raised as a result of infection and the usefulness of insulin may be reduced until the infection is controlled.

It is difficult to draw conclusions concerning the results of operation for gangrene of the extremities occurring in diabetics, for the character and severity of the diabetes and the presence or absence of infection are not infrequently not considered sufficiently.

The mortality is relatively high, however, because of the age of the patients, complications involving other organs and the presence of infection. The mortality in some series has been as high as 20 per cent, even higher. With the introduction of insulin and the more careful preparation of the diabetic patient, this mortality should be considerably reduced.

In discussing the operative procedure which should be considered, anesthesia should be considered first. Ideas have changed from time to time and differ at the present time as to the choice of the anesthetic in diabetic patients. Ether, Berkman states, is still the anesthetic of choice in the Mayo Clinic. Fitz believes that it has decidedly bad effects.

Ether, in addition to its anesthetic action, has an immediate and definite effect upon the body fats and the liver. Following administration of ether to normal dogs a marked concentration of blood-fat is noted. An increased solubility of fat of the tissues occurs in the blood-ether mixture. In dogs, made diabetic by phloridzin, ether produces an immediate hyperglycemia. Glycogen is broken down under asphyxia and transformed into sugar.

Experimentally, ether causes a temporary suspension in the excretion in the urine of nitrogen, glucose and acetone bodies, there being but little output of these substances for several hours. It has been demonstrated that the same changes may occur in patients. The blood-ether mixture seems to dissolve considerable amounts of fat from the tissues, causing a hyperlipemia. At the same time ether appears to transform glycogen into sugar, with a resultant hyperglycemia. The delicate equilibrium which is necessary to life is no longer maintained and death results from acidosis. Fat set free so rapidly is turned into acetone bodies and the carbohydrate necessary to burn it is not available. The polyuria which assists in washing away these bodies is lacking.

Nitrous oxide has been suggested as a suitable anesthetic in these cases. The cyanosis apt to be associated with this anesthetic, and the increase in blood-pressure are bad. Many of these patients already have a high blood-pressure and the arterial changes are marked. Ethylene, among the general anesthetics, is the best in these cases, for it apparently has but little effect upon sugar metabolism, is not associated with cyanosis and has but little, if any, effect upon the blood-pressure.

Spinal anesthesia is indicated for amputation in most of these cases, for it has little, if any, effect upon metabolism and has no

effect upon the general condition, aside from the headache and nausea which is sometimes associated with it.

I believe that the amputation of choice is the Carden transcondyloid amputation. It can be easily and rapidly performed and gives a good stump. Care must be exercised to not leave too much of the tendinous portion of the quadriceps extensor in the flap. Because of the impoverished blood supply, some of the tendon, if too much is left, may die and this leaves an open wound which may require a second operation.

DISCUSSION

Dr. George P. Müller: I think Dr. Lewis' point in regard to anesthesia was well taken. I use stovaine spinal anesthesia for the amputations and ethylene now invarial ly for carbuncle; of course, avoiding the cautery excision of the carbuncle. The knife excision of the mass of dead tissue will accomplish the same amount of good. In those cases of diabetes associated with gall-stone disease, chronic appendicitis, etc., we feel there is no reason to fear anything provided you recognize the constitutional condition and use proper preliminary treatment to avoid serious complications.

Dr. George M. Dorrance: I also was very much surprised to see the arterial capillary circulation as shown by Dr. Lewis. I had hoped he would mention the effect of involvement of the sympathetic nerves. Sympathectomy has been done several times, hoping to have a good effect. It has had no effect. Local anesthesia is a very simple matter and I use it whenever it is possible. I was also surprised to learn that in diabetic gangrene, particularly if the joint is involved, that he did not amputate above the knee. My present method is not to amputate below the knee, my idea being that radical operation—thigh amputation—is really conservative. No mention is made of tourniquet in operation upon diabetic gangrene, and it is universally accepted now that you do not want to make any pressure on the prominent arteries with a tourniquet. I never operate upon one of these cases unless my medical colleague concurs. I depend upon his judgment as to whether or not the patient will stand the operation.

Dr. Edward S. Dillon: I think that one thing which ought to be stressed, from the point of view of treating the diabetes, is that whereas the carbohydrate tolerance in the uncomplicated case is fairly constant from day to day, as soon as a diabetic gets an infection the carbohydrate tolerance may change with tremendous rapidity. The severity of a case varies in inverse proportion to the ability of the body to utilize carbohydrate, which presumably runs parallel with the ability of the islets of the pancreas to produce insulin. In the presence of an infection this ability diminishes

rapidly, so that in the course of a few days diabetes which may have been mild may become very severe. After all, the main problem of treating diabetes is to properly balance the food burned, both exogenous and endogenous, with insulin, that produced by the islets plus the amount it may be necessary to give by injection. In an uncomplicated diabetic these factors are fairly constant, but as soon as a diabetic becomes infected they are variable. Due to the increased metabolism more food is burned, and at the same time less insulin is produced by the islets. To avoid acidosis, therefore, it is often necessary to give rapidly increasing doses of insulin. In like manner, when the infection clears up, it is necessary to constantly watch the increasing carbohydrate tolerance and reduce the insulin dosage accordingly, in order to avoid insulin shock.

Dr. Müller: I want to answer Dr. Dorrance. I have done ten arterial sympathectomies and it did not do any good. I have never seen it do any good in the senile cases. I have practically abandoned it.

Dr. John H. Gibbon: One of the most important things about the treatment of local lesions in diabetes is the avoidance of half-way surgical measures and of the use of irritating antiseptics. Taking, for instance, diabetic gangrene of a toe, it should be generally understood that amputation of the toe alone is distinctly a surgical error, and the application of irritating antiseptics in these cases is very apt to cause a spread of the gangrene. The best treatment is simply to keep the part clean and absolutely dry. The drying process can be easily kept up by an ordinary electric light. With the proper treatment of the diabetic condition the gangrene of the toe is very apt to become limited, a line of demarcation forms and the toe can be allowed to come away itself or be simply removed with scissors. No cutting of the vital tissue should ever be done. This conservative method and the radical high amputation are the only two I believe applicable in these cases.

The College is certainly very much indebted to Dr. Lewis for his interesting presentation of the subject. I agree with Dr. Müller that Leriche's periarterial sympathectomy should not be employed in diabetic gangrene or in Berger's disease, but it must be remembered that Leriche never suggested its use in these cases. I am sure that it has a very distinct field in certain trophic lesions. I have two patients living now, more than two years after operation, carrying on their work, who but for this operation, I am sure, would have died or lost their limbs. It is a great mistake to judge the operation by the results obtained in conditions for which it was never recommended.

Dr. Lewis (closing): In cases of infection I am sometimes doubtful how to proceed, whether radically or conservatively, and I think that is where we must rely on the medical man. Dr. Dillon spoke of the changes from day to day in the diabetic, and it is important to appreciate this. As far as periarterial sympathectomy is concerned, I do not see how this procedure can have very much effect on the arterial disease. When ampu-

tation is done I do not think there is any doubt the amputation should be done above the knee-joint, and any amputation lower than that merely subjects the patient to two operations. I do not like local anesthesia for these amputations, because I think you can do it much better under ethylene. As far as I know, ethylene does not have any effect upon the diabetic patient. I think that blood chemistry studies have not been made on the diabetic patients at this time, but they ought to be made. It is not necessary to use a tourniquet in these amputations.

DIPHTHERIA AND SCARLET FEVER*

By WILLIAM H. PARK, M.D.

DIRECTOR OF THE BUREAU OF LABORATORIES OF THE DEPARTMENT OF HEALTH, NEW YORK

In this address before the College of Physicians of Philadelphia, I have been asked to touch on two topics; first the experience of New York City in the development of the drive to eliminate diphtheria through toxin-antitoxin, and second, our experience with the Dick test, the use of scarlet fever toxin in immunization and of antitoxin in treatment.

The Drive Against Diphtheria

The property of diphtheria toxin-antitoxin injections to produce active immunity in animals was known as early as 1894. It was used practically by me in 1897 in the preliminary treatment of horses to be used for the production of diphtheria antitoxin. It was not until May, 1913, that the results of its use in human beings were published.

Behring and his coworkers demonstrated that the injections were harmless in a number of children. With this knowledge I determined to undertake a thorough study of the problem of the prevention of diphtheria by active immunization. There were a number of questions to be answered before an estimate of the value of active immunization against diphtheria could be made. These were the percentage of children who were immune at the different ages as determined by the Schick test, the percentage of nonimmunes who became immune after one, two and three injections, and the duration of the immunity.

From the beginning I was so fortunate as to have Dr. Zingher associated with me in this work, and later Dr. Schroder. At first we used 4 L+ doses of toxin suitably neutralized by antitoxin in each dose. The preparation of the toxin-antitoxin was later modified by me to the now generally used one containing in each injection one-tenth of an L+ dose. This gives less local protein

* Read April 7, 1926.

reaction and has shown itself incapable of harmful changes and has been safely used in hundreds of thousands of children. We have lately used in several thousand children toxin reduced by contact with formalin to toxoid. The earliest tests were done in children's institutions, where the children usually remained for several years. As soon as the results showed that the immunity persisted for at least three years, work was started in the baby health stations and in a few schools. While it was well known that the majority of the cases of diphtheria and nearly all the deaths occurred in children under the age of five years, it was determined to make the first general effort for preventive work in the schools, because of the great difficulty in getting access to the children of preschool age. Further, the diphtheria prevention work in the schools is a valuable preliminary step to any attempt to immunize the children of preschool age. Through the schools, the parents will soon learn to appreciate the value and the harmlessness of these newer measures of diphtheria prevention. Many homes can thus be reached in a short time. The consent blanks and other literature taken home by the children tell the parents all about the work. The prestige of the school is behind this work, and the parents have to take a definite action by either giving consent or refusing to do so. We found in our school work that the wholehearted coöperation of the principal and teachers was of greatest importance to obtain successful results in a school. In some of the schools we were thus able to obtain consent from fully 60 to 80 per cent of the parents.

From the beginning of the work we had the wholehearted coöperation of the superintendent of schools, Dr. Ettinger, and, when he retired, of his successor, Dr. O'Shea. Our method of operating was as follows: Having personally obtained the approval of the school principal as to the date of the school visit, the teachers were instructed as to the value and harmlessness of the treatment. A sufficient number of consent slips were then left with them. The next day each child was given a slip and was required to take it home to its parents and to bring it back with the consent or refusal of the parents to have the immunization done. The following circular is an example of the consent slip:

To Parents and Guardians. *Do you realize the danger to your children from diphtheria?* From January 1, 1924, to December 31, 1924, there were in the City of New York 8244 cases of *diphtheria* with 714 deaths. These 714 deaths occurred because the *diphtheria* was not discovered early enough to give antitoxin promptly. Over 66 per cent of the younger and about

33 per cent of the older school children are susceptible and may catch this disease.

Your child should be protected against diphtheria now by the new method of vaccination.

It can be found out by a simple test whether your child is safe or needs the vaccine. This test is free from all danger, it may be administered by your family doctor and it will not make your child sick. It requires the placing into the skin or beneath the skin of the test liquid.

If the test shows that your child is safe, nothing further need be done. If it shows that the child may catch the disease, ask your family doctor to give the treatment. Three injections of vaccine given at weekly intervals will almost surely give a lifelong protection.

The test and the vaccine are harmless, no matter how young the child. The only possible objection is that the spot on the arm where the vaccine was injected may become red and painful for one or two days. The vaccination can be given at any season of the year.

About six months after the giving of the vaccine a second test is usually made to find out whether immunity has been fully established. A certificate is then issued to show that the child is protected and safe as far as catching this disease is concerned.

Because *diphtheria* destroys the lives of so many children and has caused the exclusion from school of over 17,000 children during 1924, the Boards of Health and Education think it wise to immunize each child.

Don't Risk Waiting! Don't Delay Going to Your Family Doctor. If you cannot obtain the services of a private physician sign the slip attached and a health department physician will apply the test and the remedy at the school. Those too young to go to school will be treated at the nearest baby health station or clinic. See telephone directory for address.

Consent for the Original or the Modified Schick Test and Immunization

I hereby request that ..... of ..... may
(Name) (Address)
receive from the Department of Health physician the test to determine if the child is liable to contract diphtheria. If found to be in danger of contracting this disease, I consent that the protective vaccination and the final immunity test be given.

Dated, New York.....192.....

....
(Parent) (Guardian)

Note. The young children who are just beginning to go to school are usually very susceptible to diphtheria and are, therefore, usually given the protective vaccination without any preliminary test.

Referred by .....

We also gave the scholars a circular of information showing the reduction in mortality and morbidity since the introduction of the work and describing the Schick test and the harmlessness of the vaccination.

Since 1922 we have been devoting our attention during the summers to the preschool children. For this purpose we utilized our baby health stations. The necessity of doing the preschool child is shown in the table giving the results in the Schick test in 4502 children by Dr. Zingher:

TABLE I.—RESULTS OF SCHICK TEST IN BABY HEALTH STATIONS AND VACATION PLAYGROUNDS (MANHATTAN AND THE BRONX)

Age.	Total.	Schick positive.	Schick negative.	Schick positive, per cent
From 6 to 7 months	53	30	23	56.6
From 7 to 8 months	41	26	15	63.4
From 8 to 9 months	62	52	10	83.8
From 9 to 10 months	58	54	4	93.1
From 10 to 11 months	61	53	8	87.0
From 11 to 12 months	34	31	3	91.1
From 1 to 3 years	1727	1438	289	83.2
From 4 to 6 years	1328	779	549	58.6
From 7 to 10 years	903	330	573	36.5
From 11 to 14 years	235	53	182	22.5
Total	4502	2846	1656	

The following circulars are some of those we are using. The first is mailed to the physician and the second given out in the schools to be taken home to the parents. Then we distributed cards at the baby health stations, day nurseries, etc., and on the completion of the injections we gave a certificate.

The following is the circular letter sent to the physicians:

IMMUNIZATION OF CHILDREN OF PRESCHOOL AGE AGAINST DIPHTHERIA.
DEPARTMENT OF HEALTH, CITY OF NEW YORK

The Department of Health is going to start an active campaign throughout the city this summer to immunize the children between six months and six years of age against diphtheria with toxin-antitoxin. This work will be carried out in the baby health stations, day nurseries and mother and babies' vacation playgrounds in the public schools. *The Department would like to suggest to the physicians throughout the city to do this work on the young children in their private practice.*

For that purpose fifty circulars and twelve diphtheria vaccination certificates similar to those enclosed will be supplied to any physician in the city who wishes to use them in connection with the immunization. *These circulars can be obtained on request from the research laboratory, foot of East Sixteenth Street, New York City.*

There is no reason why every young child should not be "vaccinated" against diphtheria just as it is vaccinated against smallpox. This is particularly true of the children most susceptible to diphtheria, that in those belonging to the age group between six months and six years.

To simplify the immunization, we suggest that the preliminary Schick test ordinarily used with older children should be omitted and that each child of this group should be given the three injections of toxin-antitoxin.

The toxin-antitoxin can be obtained free at certain selected Department of Health stations. This list has been previously published in the weekly bulletin, but will be republished in a short time. It is also obtainable at the borough offices of the Health Department and at the Research Laboratory, foot of East Sixteenth Street, New York City.

Diphtheria Prevention Department, Research Laboratory, Foot of East Sixteenth Street, New York City.

Please send me fifty circulars and twelve diphtheria vaccination certificates.

Name

Address

Circular Emphasizing Dangers of Diphtheria for Young Children, Somewhat Similar to the one for School Children

Vaccination Against Diphtheria

Department of Health, City of New York

Parents! Guardians! Vaccination against smallpox was a wonderful discovery. Millions of lives have been saved by it. You had your children vaccinated to keep them from getting smallpox. *Now science has won another great victory! We can now vaccinate your children against diphtheria*, so that they are protected against this terrible disease!

Save your children from diphtheria. This is no longer an experiment; it is a certainty! More than 200,000 school children have been protected against diphtheria during the past two years in the public schools of New York City.

This is how it is done: First, the children are given a very small injection into the skin, called the Schick test, to find out if they are liable to catch diphtheria. Second, those that are found to be liable to catch it are given three injections of a different kind which protects them for many years and probably for life.

Young Children Catch Diphtheria Most Easily. Your children who are too young to go to school need protection against diphtheria much more than school children. Young children between six months and six years of age are especially liable to catch diphtheria and frequently have it in its worst form. Out of every 100 children who get diphtheria, 85 are less than five years of age. These young children are therefore given the protective vaccination injections without any preliminary Schick test.

Diphtheria Protection is Absolutely Safe. One of the fine things about this method of protection against diphtheria is that *young children are not made sick by it.* The younger children are, the better they take it. Don't wait until your children go to school! Have them vaccinated at once!

Diphtheria is a Dangerous Disease and Kills Many Children! Diphtheria is a treacherous disease. It not only kills many children, but even when they recover many of them are left crippled with heart disease for years if not for life. Nor is the child always protected against diphtheria after one attack.

New York City is Conquering Diphtheria by the New Method. Since the new prevention methods have been extensively employed in this city, the number of deaths from diphtheria has been reduced from 1239 in the year 1919 to 891 during 1921. Will you not help reduce the number still further?

Will you Help to Wipe out Diphtheria? Don't keep this to yourself! If your neighbor's children are too young to go to school, be sure to show them this circular. You may help save other lives!

Children are now Vaccinated against Diphtheria at the baby health stations. If you cannot afford the services of a private physician, the Department of Health will give them diphtheria protection at the nearest baby health station. Ask the nurse at the baby health station when the doctor will be there to vaccinate the children.

Get a Diphtheria Certificate for Your Child. A diphtheria vaccination certificate will be given to each child after it has received the three protective injections. This certificate will be of value to your child when beginning school. If at that time a Schick test shows that your child is surely protected against diphtheria, a special diphtheria protection certificate will be given.

BABY HEALTH STATION

Important Official Notice.

DIPHTHERIA PREVENTION

Department of Health — City of New York

Protect your young children against Diphtheria! Next week the doctor will vaccinate against Diphtheria the children of the Baby Health Station and also all those who are too young to go to school. Be sure to ask the nurse about this wonderful opportunity as soon as you receive this card. She will give you all the information you need. Also tell your neighbors who have young children about it. These injections may save your children's lives!

This is a special opportunity which the Department of Health offers to you. Will you not take advantage of it?

The doctor will give the protective injections on.....
during the month of.....

DEPARTMENT OF HEALTH—CITY OF NEW YORK

DIPHTHERIA VACCINATION CERTIFICATE

This is to certify that.....Age.....
residing at.....has receive the three protective injections of toxin-antitoxin against DIPHTHERIA. The final DIPHTHERIA PROTECTION CERTIFICATE will be issued when the child is given a Schick test after admission to school and shows a negative Schick reaction.

ISSUED BY.....M.D.,
Medical Inspector.

ROYAL S. COPELAND, M.D.,
DATE..... Commissioner.

In 1927 we expect to make a more earnest effort to get all the preschool children. On January 1 we turn over the force doing the diphtheria preventive work from the Bureau of Laboratories to the Bureau of Child Hygiene. This will make it easier to get at the preschool child. We are also trying in every way to impress the physicians of the city that it is their responsibility to urge their patients to either let them immunize their children for a very moderate fee or to allow the health officials to do so. The various borough county medical societies have promised to aid in the movement and we expect results from this coöperation next year.

Until recently we have given the preschool children the three injections of toxin-antitoxin without a previous Schick test. The school children, on the other hand, were first given the Schick test, and then only those who on the third or fourth day showed the positive reaction were given the three injections at intervals of one week. In New York City about one-half of the children are already immune when they reach the school age, so that the doing of the Schick test actually saves trouble. Lately we have been using the first injection of the toxin-antitoxin for both a diagnostic and immunizing purposes. Toxin-antitoxin gives its best results when it is slightly underneutralized. One cubic centimeter should kill the majority of guinea-pigs within one month and 5 cc. should kill in from seven to ten days. If it is of this strength it can be used either intracutaneously or subcutaneously as a Schick test. We have therefore, injected 1 cc. subcutaneously just above the bend of the elbow and read the reaction on the fifth or sixth day. Those who were positive to the slightest degree received a second injection and those who were distinctly positive a third injection. We are now using the toxin-antitoxin in this way in more than one-half of the first injections and soon intend to use it in this way in all. It is absolutely essential that the toxin-antitoxin be standardized as to its toxicity if it is to be used in this way and given just beneath the skin.

For the present at least we intend to use toxin-antitoxin instead of toxoid for immunizing purposes. The results are equal, so far as immunizing results are concerned, and after most careful investigation we believe the slight sensitization which the minute amount of serum produces in some is of no practical importance. In time, when the toxoid has been standardized, it may be wise to utilize it.

The Results Accomplished. A great city like New York cannot expect to have the brilliant results obtained by such cities as

Auburn, in New York State, and New Haven, in Connecticut, for it is impossible to make such a personal and intensive drive. This we can say, however, that the experimental use was done so carefully and merged so gradually into its general preventive use that there has never been any organized opposition, and that the example set by New York City has spread and been copied more or less generally throughout the United States. Even in New York City the results are impressive.

Between 1918 and 1922 we were gradually enlarging the diphtheria preventive work. In 1922, however, we received a grant of money from the New York chapter of the Red Cross Society and developed the school work as it is now carried on. We were able to appoint six physicians, six nurses and six helpers.

In 1923 we obtained a budget from the city to establish the work on a permanent footing. This was while Dr. Copeland was Commissioner of Health. We have now tested and, when indicated, immunized nearly 750,000 children. The following figures are certainly striking:

Diphtheria, Incidence and Mortality, 1921–1926, in New York City

Year.	Cases.	Death.
1921	15,110	891
1922	10,427	874
1923	8,050	547
1925	9,051	663
1926	7,531*	477*

* Figures in address for first quarter of year only. These for the full year have been inserted as address was being put in print.

No one who has studied the problem of diphtheria would assert that this improvement is due wholly to toxin-antitoxin immunization. With this has gone instruction in the need of the early reporting of suspicious cases of diphtheria and the urgent necessity of giving antitoxin promptly and adequately.

These figures are encouraging, however, and should aid in getting all interested in eliminating diphtheria to help in every way the drives now being made in different parts of the country.

Scarlet Fever—its Prevention and Serum Treatment

The majority of physicians believe that our knowledge concerning the etiology, prevention and cure of scarlet fever began with the discoveries of the Dicks and Dochez. A careful study of the literature reveals the fact that an immense amount of fruitful bacterio-

logical investigation started after Loeffler, in 1884, drew attention to the presence of the hemolytic streptococci in the throats of persons sick with scarlet fever. The bacteriological findings led to the development of a practical therapeutic serum by Moser and of an efficient vaccine by Gabritschewsky. Before taking up the present status of the prevention and specific treatment of scarlet fever we will do well to consider briefly the results achieved by them.

In 1902, Moser believed that the evidence of the relation of these streptococci to scarlet fever was definite enough to justify his trying to produce in horses a therapeutic serum. His method of obtaining the cultures was founded on the assumption that if these micro-organisms were the cause of scarlet fever that he would probably obtain them in pure culture from the blood of early severe cases. He, therefore, made blood cultures from a number of fatal cases of scarlet fever with positive results and injected increasing doses of a number of these streptococci with the broth in which they had grown, subcutaneously into horses. In this way he would use any toxin that was in the broth as well as the bacteria. He found that after giving increasing amounts for a number of months that some of the horses produced a serum which had remarkable effects when given to human beings suffering from scarlet fever. He used large doses of 100 cc. and even more. Each case received during the first forty-eight hours several hundred cubic centimeters. The results following the injection of serum from the best horses were striking when the serum was given early. The toxic symptoms began to abate within a few hours. Within twelve hours the rash was much less, the mind clear, the appetite better and the temperature was down from 3° to 4°. These results were so striking that Moser and others who used the serum believed that it must be specifically antitoxic and also bactericidal, and that the results were strong even if not conclusive evidence that these streptococci were the cause of the disease itself rather than of a constantly present complication.

In 1906, Gabritschewsky attempted to immunize children against scarlet fever by giving them both toxin and the killed culture of streptococci. The culture after four days of growth was killed by carbolic acid and centrifuged, so that in 1 cc. of toxic broth there was about $\frac{5}{100}$ of a gram of organism. One-half cubic centimeter of this toxic fluid plus the organisms when injected into children produced in about 15 per cent a rash similar to that of

scarlet fever, and in about 2 per cent to the rash were added all the other symptoms of a moderate case of scarlet fever. The majority of the other children showed a local inflammatory reaction, while others showed no reaction at all. Among these were those who had had scarlet fever. A similar vaccine prepared from streptococci obtained from erysipelas cases tested in thirty children did not produce this generalized rash but only a local reaction. He found that when a second dose, double in amount that of the first, was given a week later, there were almost no cases of generalized rash, and that on the third injection, even though the amount was again doubled, there were practically no rashes. This lack of reaction to the second and third doses he believed to be due to a beginning immunization.

At the same time that Gabritschewsky published his work, a Russian colleague, Savchenko, showed that the broth culture gave a toxic filtrate and he utilized this in horses. He first gave injections of filtrate alone, and then, after antitoxin had developed, the streptococci were added. His idea was that scarlet fever being both a toxemia and a septic infection, a serum to be fully effective should possess both antitoxic and bactericidal qualities. Gabritschewsky's vaccine was used in a great many thousand of Russian children, and those who used the vaccine and made reports gave very favorable results of its immunizing qualities.

The next striking addition to our knowledge was in the discovery, by Schultz and Charlton, that the convalescent scarlet-fever serum when injected intracutaneously into the skin of a patient suffering from scarlet fever caused a blanching of the exantheme. Even moderate dilutions of the serum would have this effect.

This leads us to the work in the United States. In 1914 one of our laboratory assistants, though an accident, drew into her mouth several cubic centimeters of a mixture of a twenty-four-hour culture of a strain of hemolytic streptococci from a case of scarlet fever and of a strain from a case of puerperal fever. This inoculation was followed, forty-eight hours later, by the development of a typical case of scarlet fever. We tried, as others had tried, to produce the disease in animals and with the same negative results. An account of this accidental production of a case of scarlet fever was published.

At the close of the war, without knowing of the progress of the work of each other, Dochez and the Dicks were both making studies on the etiology and specific treatment of scarlet fever.

Dochez began by using the filtrates of scarlet-fever cultures and injecting them subcutaneously into horses, but he was unable to obtain a potent antitoxic serum, and so he turned to a method which theoretically he thought might give better results. He injected subcutaneously into horses small masses of melted nutrient agar, and then, into this, living scarlet-fever streptococci. This led in the majority of horses to the development of small abscesses. When these healed the horses were again injected, but now with a larger quantity of the living streptococci in melted agar. After seven or eight months he was gratified to learn that this serum possessed blanching qualities of even greater potency than that from the average convalescent case of scarlet fever. This serum, used at first by Blake in New Haven and later by me in the contagious hospitals of the Department of Health of the City of New York, showed striking therapeutic effects. These results were similar to those already reported by Moser, Schick and others with the Moser antiscarlatinal horse serum.

At the same time that Dochez was working on the antitoxic serum, the Dicks added to the demonstration that the streptococci were the cause of scarlet fever the important evidence that three persons developed scarlet fever after the intentional inoculation of the living scarlet-fever streptococci in the throat. They also demonstrated that this toxin discovered by Gabritschewsky and Savchenko had the property in high dilution of producing a reaction in the skin which could be used for determining immunity similarly to that of the diphtheria toxin when used in the Schick test. By very careful research they developed that an amount of toxin which when injected intracutaneoulsy in nonimmunes in an amount of $\frac{1}{10}$ cc. would develop a reaction of about 2 cm. in diameter was an amount which would differentiate between persons who were susceptible and those who were insusceptible. Those that developed reactions of less than 1 cm. were believed to be protected against scarlet fever.

During the past three years the work of the Dicks and Dochez have so impressed American investigators that the use of the serum therapeutically and of the streptococcus toxin in the Dick test for immunization have made great progress.

The Dick Test. The technic used in making the Dick test is identical with that used in the Schick test except that it is read at the end of twelve to twenty-four hours. The standardization of

the toxin is more difficult because, up to the present time, only human beings and a special strain of Swiss goats are suitable for testing, and, as both persons and goats differ among themselves in their susceptibility, it is necessary to test on ten or more individuals to get an accurate result. Just as there are pseudoreactors to the diphtheria toxic broth, there are to the scarlet-fever toxic broth. While in the case of diphtheria there is a difference in the time of the development of the true and pseudoreactions, there is none between the true and pseudoreactions to the scarlet-fever specific toxin and the less specific endotoxin and nonspecific foreign protein. The advantage in doing a control test is that if there is no pseudoreaction, with it one is confident that there is no antitoxic immunity if the Dick test is positive. Just as in diphtheria a person may be temporarily immune because of possessing anti-bacterial substances, even if there is no antitoxin. We have now sufficient experience to affirm there is no doubt that those who have a negative Dick test are generally immune when exposed to scarlet fever, while those who have a positive test frequently contract the contagion. A few examples have occurred in which children who gave a negative Dick test have later developed scarlet fever. This either means, if a proper technic was used, that the present standard doze of the Dick toxin is not sufficient or that the immunity disappears. We have for some time been using double the strength of the standard Dick dose of toxin and have not in a rather small number of cases found any to develop scarlet fever who gave a negative Dick test with this amount of toxin. This change has caused only a moderate increase in the positive cases. The scarlet-fever toxin, although fairly stable, does at times rather quickly deteriorate, so that some of the supposedly negative Dick tests are really the result of insufficient doses of toxin. The giving of double the standard dose for the Dick test not only is probably a more reliable procedure when the toxin is of full strength, but allows for this unexpected deterioration when it happens. The toxin used as a control for pseudoreactors should be boiled for at least two hours, as it is very resistant. It is possible that a fifty per cent excess would be sufficient,

The Percentage of Immune at Different Ages. The interesting fact has been brought out that at the different age groups about the same proportion of children give a negative reaction to the Schick and the Dick tests. This does not necessarily mean that the same children are negative or positive to both tests.

The following table, from Zingher, gives an average picture in the children of New York City:

TABLE II. THE DICK TEST AT DIFFERENT AGE GROUPS

Age	Per cent. Dick positive.	Age.	Per cent. Dick positive.
0 to 6 months	44.8	4 to 5 years	48.4
6 to 12 months	65.3	5 to 10 years	33.6
1 to 2 years	71.6	10 to 15 years	22.8
2 to 3 years	64.2	15 to 20 years	16.8
3 to 4 years	60.5	20 years up	14.4
		Total	29.2

We have found the same striking difference in the percentage giving the reaction to the Dick test between city and country children and between children in private schools and public schools as to the Schick test. Thus Zingher found 83 per cent positive in two private schools and 18 per cent positive in homes for destitute or friendless city children of about the same age.

THE TRANSMISSION OF IMMUNITY BY MOTHERS TO THEIR OFFSPRING. Zingher found the same relation between immunity in mothers and their infants as in the case of diphtheria. Immune mothers give birth to immune infants, and nonimmune mothers to nonimmune infants. At five months babies immune at birth begin to lose it and at eight months may be definitely positive to the Dick test.

THE DICK TEST IN ACUTE AND CONVALESCENT CASES OF SCARLET FEVER. The work of the Dicks, Trask and of others suggested to Zingher a careful test of this reaction in the scarlet-fever patients at the Willard Parker Hospital. He also used the Schultz-Charlton test. The results were as follows:

TABLE III.—THE DICK TEST AND SCHULTZ-CHARLTON PHENOMENON IN SCARLET FEVER

Test.	Days of illness. 1–4 days.	5–7 days.	8–10 days.	11–15 days.
Dick test	+ or ±	+ or ± or ±	± or −	−
Schultz-Charlton	−	− or ±	± or +	+

+ with Dick test = reaction. + with S.-C. test = blanching of rash of another case.

These tests may be used for diagnostic purposes within limitations. A definitely negative Dick reaction during the first three days of a case of suspected scarlet fever makes the diagnosis doubtful, and the absence of the Schultz-Charlton blanching phenomenon when $\frac{3}{4}$ cc. of a proper dilution of a highly potent antitoxic serum is injected into the patient's skin has a similar import. At the present time we believe that a certain number of cases of scarlet fever are

due to streptococci of other strains than those commonly met with. These produce different toxins and are not neutralized by the ordinary antitoxin. Some of the cases of septic sore-throat and wound infections with scarlet rashes are possible due to such strains. These cases make the results of the Dick and Schultz-Charlton tests less definite. Until these cases have been subjected to further study they must be considered as scarlet fever. Later they may be given a separate name such as parascarlet fever. The toxins and agglutinins produced by the ordinary strains of streptococci from scarlet fever cases vary in minor details, as shown by Williams, myself and others, so that it is not surprising that some cultures vary more radically.

Active Immunization. Earlier in this paper we alluded to the work of Gabritschewsky and his followers. With the Dick test the dosage can be standardized and the immediate and later immunity results determined by retesting from time to time. The scarlet-fever toxin is much less injurious to the local tissues than the diphtheria toxin, so that there is no need of adding antitoxin to it. It is possible that a toxoid may be developed which will be better than the toxin. The most suitable dose of toxin varies with conditions. If scarlet fever is prevalent, or if there has been contact with a case, it is very desirable not to give sufficient toxin to make a rash in any of the very susceptible children, for there is no certain way of determining in such a case whether it is a case of light scarlet fever or of a toxin rash. If, however, no contact with scarlet fever is possible, the development of a slight scarlet rash following the toxin injections is of less importance and larger doses can be given. The Dicks give a very interesting table of the relation of the total skin doses to the immunity results:

Table IV.—Direct Relation between the Amount of Toxin Administered and the Immunity Obtained (Dick and Dick).

Total amount of toxin administered, expressed in skin-test doses.	Completely immunized: skin test negative, per cent.	Not completely immunized: skin test positive, per cent.
From 100 to 1,000	0.0	100.0
" 1,000 to 2,000	14.3	83.7
" 2,000 to 3,000	21.7	78.3
" 3,000 to 4,000	41.9	58.1
" 4,000 to 5,000	45.7	54.3
" 5,000 to 6,000	66.0	34.0
" 6,000 to 7,000	59.6	40.4
" 7,000 to 8,000	57.1	42.9
" 8,000 to 9,000	55.3	44.7
" 9,000 to 10,000	72.7	27.3
" 10,000 to 12,500	91.8	8.2

In this table the Dicks do not give the number of injections. There were probably at least four in the larger doses. If the amounts given up to 3000 skin doses had been given in 3 doses, the percentages of immunity would surely have been much better than those given. The following tables show the results in our tests following the injection of 1600 to 10,500 skin doses. The tests in these tables were made by Schroder and read by her and myself.

TABLE V.—RESULTS OF INJECTING 200, 400 AND 1,000 SKIN-TEST DOSES AS SHOWN BY RETEST FIVE AND THIRTEEN MONTHS AFTERWARD AT ST. DOMONICKS.

Original retest at 5 months.			Per cent.	13 months, per cent.
One hundred positive cases of St. Dominicks test by Dr. Schroder	++	= 0		++ = 20 very susceptible
	+	= 8	= 9.8	+ = 9 susceptible
	±	= 27	= 33.3	± = 14 doubtful
	−	= 46	= 56.8	− = 57 immune
	* Total	81		Total 100

* Some of the children were away at the first retest.

TABLE VI.—RESULTS IN DICK POSITIVE CHILDREN THREE AND A HALF MONTHS AFTER RECEIVING THREE IMMUNIZING INJECTIONS OF APPROXIMATELY 1000, 2000 AND 4000 SKIN DOSES.

	Reactions three and a half months later	
(Strongly positive)	1 = +	9% probable susceptible
11++or++1	3 = ±	27% doubtful
	7 = −	63% immune
10+or+1	1 = ±	10% doubtful
(Moderate reactors)	9 = −	90% immune

TABLE VII.—RESULTS OF FIVE IMMUNIZING INJECTIONS AT THE END OF THREE AND A HALF MONTHS APPROXIMATELY 500, 1000, 2000, 3000 AND 4000 SKIN-TEST DOSES.

	After three and a half months	
	1 = +	2% probable susceptible
32++or++1	5 = ±	= 17% probable immune
Strongly positive cases	23 = −	= 81% immune
	*3 = ± negative pseudo	
37+or+1	34 = −	
Moderate reactors	*1 = + negative pseudo	100% immune
	*2 = ± negative pseudo	
	1 = +	1% probable susceptible
All positive reactors	5 = ±	7% probable immune
69+to++1	57 = −	91% immune
	1 = + negative pseudo	
	5 = ± negative pseudo	

* 1 + and *5 + are considered negative pseudo reactions because of an equal reaction in the control neutralized by convalescent serum.

The persistence of the acquired immunity following injections remains in doubt. With small injections, such as the cases recorded in Table VI, many of the originally very positive cases have after a year relapsed, while the originally moderately positive cases have generally continued to hold their immunity or to increase it. With larger injections, such as those recorded in Tables VII it is hoped that the marked positive cases will not only develop immunity but hold it for at least a year or two. These have held it now for fifteen months.

We note in these tables that the moderately reacting cases develop complete immunity much more readily than the strongly reacting cases. Again that the response is quicker than after diphtheria toxin-antitoxin or toxoid injections. This corroborates the views of Gabritschewsky and the statements of the Dicks that immunity develops in many within one or two weeks.

The results of a second series of injections in those who have not responded to the first series have been shown by Zingher to have been excellent.

The *persistence* of a negative Dick reaction in those naturally negative. Observations on several thousand children by Schroder and by Zingher at intervals of time up to thirteen months have shown that a naturally acquired immunity is persistent for at least one and a half years. Only about 2 per cent have been found to change. It is fair to assume therefore, that as in the case of naturally acquired immunity to diphtheria the immunity to scarlet fever naturally acquired is apt to remain. This finding, which is in accord with the findings of the Dicks, encourages us to hope that, with larger and perhaps more numerous doses, even the very positive cases may acquire such a high degree of immunity that it will last for at least a few years. We already have the knowledge that these immunizing injections are very useful in stopping an outbreak.

The Prevention of Scarlet Fever in those that are Exposed. We have in the scarlet-fever antitoxin a fairly certain preventive against the development of scarlet fever, but it is not as certain as a dose of diphtheria antitoxin against the development of diphtheria, possibly because, as already stated, under scarlet fever we include a small percentage of cases due to other than the usual strains of scarlet-fever streptococci. Our most definite experience was when we tested it in 40 consecutive children who had been definitely exposed in families and but 1 of these developed scarlet fever; this was a mild and rather atypical case. At the

same time, in 100 other children equally exposed, to whose parents we offered the injection, the serum was refused, 9 of these children developed scarlet fever. On the other hand, a very considerable percentage of those receiving the preventive inoculations developed more or less severe rashes, and a few also developed some of the other accompaniments of serum sickness. It was an interesting fact that the severity of these reactions averaged the same in children who previously had had toxin-antitoxin and in those who had not had the injections. Because of the frequent serum sickness and the short duration of the immunity given by the serum, it has been my policy not to inject serum in cases that have been in contact with scarlet fever and show no symptoms whatever, but rather to do a Dick test and immediately begin the immunization with vaccine in the positive cases. Within twelve hours the Dick test can be read, and, if either then or later a sore throat develops in a positively reacting child, a therapeutic dose of antiscarlatinal serum is given, which will generally prevent the development of symptoms and of a rash and will certainly prevent the development of a severe infection. Otherwise the 4 injections of vaccine are given.

The Serum and its Therapeutic Use. Horses injected with the toxin produce antitoxin, but not antibacterial antibodies. Those injected with both toxin and streptococci produce both antitoxin and bacterial substances. Those injected, as suggested by Dochez, with melted agar impregnated with live organisms produce first a strong antitoxin and later a considerable amount of antibacterial substance. Blake has pointed out that scarlet fever is, first, a toxemia, then a mixture of toxemia and sepsis, and if complication persist after the blanching of the rash a sepsis only. When given early, the good results are mainly due to the antitoxin. We have had strikingly favorable results later in the disease with serum which was both antitoxic and bactericidal. The scarlet-fever antitoxin is of the same quality whether made by the Dochez method, by the Moser method, or by the modification of Savchenko's method used by the Dicks. In almost every case of scarlet fever in which the rash is still well developed, a sufficient dose of antitoxin will cause a rapid clearing up of the toxic symptoms. If the serum is given intravenously and in sufficient amount the changes are so rapid as to be almost startling. A patient who is delirious, with a temperature of 104° or 105°, and retains no food in his stomach, will in the course of two or three hours rapidly improve, and in the course of six hours his temperature will be nearly normal,

his rash will be much less pronounced, his mind will be clear, the pain in his throat will be largely gone and his whole appearance will be that of one who has progressed to convalescence. Sufficiently large intramuscular injections will be followed by similar results, but they will not appear so rapidly; the six hours will be lengthened to twelve or eighteen hours before the full benefits have been accomplished. There are a very few exceptional cases in which only a little improvement occurs. We have at the present time a great advantage over Moser in that we can test the potency of a serum before using it and so use only a potent preparation of antitoxin.

The Dosage. At the present time, however, the scarlet-fever antitoxin cannot be as accurately standardized as the diphtheria antitoxin. There are two methods of judging the potency. One is by the blanching effect. The dilution of the antitoxic serum which, when $\frac{3}{4}$ cc. are injected intradermally, still gives a blanching effect in the reddened skin of a scarlet-fever patient is noted. The other is the estimation of the amount of Dick skin-test doses of toxin which 1 cc. of the serum will neutralize when tested in the skin of a susceptible person, $\frac{1}{10}$ cc. of the mixture being injected. According to the partial or absolute neutralization of the toxin by the body would be the reaction of the mixture of toxin and antitoxin in the skin. At the present time it is doubtful whether the potency of the scarlatinal antitoxin tested on one or two subjects either by the Dick method or by the Schultz-Charlton method will give results which are within 30 per cent of accuracy. Of course, when a large number of tests are made, this accuracy is much increased, but it is difficult to get children enough or scarlet-fever patients enough to make a large number of tests on a given lot of serum. Notwithstanding these difficulties the serum can be standardized sufficiently well for all practical purposes. According to the method used the serum strength is estimated in the number of skin-test doses that are neutralized or in the dilution of serum which blanches. A number of us have agreed to consider as a unit the amount which will neutralize 100 Dick skin-test doses. The Dicks believe that the results should be read at the end of forty-eight hours. It is hoped that very soon we can agree upon a unit which will represent the same thing when tested by either of these two methods. For an ordinary case of scarlet fever 5000 units, which is an amount of antitoxin which will neutralize 500,000 skin-test doses, will be sufficient to bring about a recovery. In very severe

and malignant cases at least four times this amount should be used. When smaller doses are given, the more severe cases are apt to relapse. The temperature falls 2° to 4° and then rises again. So far as complications are concerned, it is believed that the early use of the antitoxin lessens the liability to complications, but after the rash has disappeared the antitoxin has little or no effect. This raises the question as to whether an antibacterial serum should be added to the antitoxic serum? It is perfectly possible to have a serum of this double potency. During the past three months we have used such a serum and the results have been remarkably good. It is difficult, however, to know what would have happened if we had used simply an antitoxic serum, as the cases differ so much from year to year in their severity and tendency to develop complications.

Conclusions. The Dick test is a reliable test for determining immunity. In the scarlet-fever toxin we have a vaccine which when injected gives a rapid immunity so that within a week or ten days at least partial immunity can be established. The duration of active immunity can be established. The duration of active immunity, although prolonged, is probably less than that which follows injections of diphtheria toxin-antitoxin, but only time can tell as to the exact duration of the immunity. At present it seems best to give 4 doses of vaccine, namely 500, 2000, 6000 and 12,000 skin-test doses. A fifth dose of 20000 will increase the duration of the immunity.

An injection of 1000 units of scarlet-fever antitoxin will give an immunity for about ten days. As a rule, it is better to give the Dick test to those who have been exposed and then to immunize the Dick-positive children with the vaccine, the antitoxin being given only on the development of a sore-throat or other symptoms.

The therapeutic value of the antitoxin is thoroughly established. It should be given as diphtheria antitoxin is given, at the earliest possible moment and in a sufficient dose. If, however, the temperature rises again, a second dose should be given.

Five thousand units is sufficient for a mild case, but at least two to four times that amount should be given a severely toxic one.

It is still to be determined whether antibacterial antibodies are of definite value and should be added to the antitoxin. In severe and toxic cases it should be given intravenously. Mild cases, with a temperature never rising above 101°, do not require serum. The frequent development of a mild or severe serum reaction about counterbalances the good done by neutralizing the toxin.

DISCUSSION

Dr. John A. Kolmer: I am sure that all here assembled join with me in expressing deep appreciation of the paper presented by Dr. Park, and particularly since the subjects discussed are commanding so much attention from the standpoint of public health. My remarks will be devoted largely to scarlet fever, because I believe that active immunization against diphtheria is practically a solved problem and largely due to the excellent work accomplished by Dr. Park and Dr. Zingher in New York City. I agree with Dr. Park that the Schick test may be dispensed with in children from two to five years of age, but with older children and adults, I believe, a preliminary Schick test is advisable in order to prevent the needless immunization of individuals who are naturally immune to diphtheria. Furthermore, the kind and degree of Schick reaction is of value in estimating the first dose of toxin-antitoxin, and particularly among adults, since a severe reaction and a pseudoreaction indicate, in my experience, that the first dose of toxin-antitoxin should be cut in half in order to reduce the severity of the local and general reaction following the injection.

Relative to active immunization against scarlet fever, I may mention in passing, with pardonable pride, the work which I did in 1912 with scarlet-fever streptococcus vaccines in the Philadelphia Hospital for Contagious Diseases. My work was inspired by the reports from Russia by Gabrickevsky and others; our results were very encouraging and I now regret that we did not continue the work at that time. In other words, we shared the opinion at that time that, while streptococci were important in scarlet fever, they were not the primary cause of the disease, and I think the Drs. Dick are to be congratulated in adhering to their work, and I hope that a fitting memorial will be erected to those who volunteered to receive injections of scarlet streptococcus toxin which resulted in furnishing the best proof so far available that this organism is the real cause of scarlet fever.

In my experience, active immunization against scarlet fever with toxin is not yet on a satisfactory basis, and largely because we are still in ignorance of the proper doses to administer. I am convinced that the first dose should never be more than 500 skin-test doses, but that an immunity is not likely to follow unless the total amount of toxin injected reaches at least 15,000 to 20,000 skin-test doses. My practice at present is to give five injections at intervals of a week, and in the doses recommended by the Scarlet-fever Committee of Chicago. If the Dick reaction is larger than a twenty-five cent piece I generally make my first dose 250 skin-test doses, in order to prevent the possibility of producing a generalized reaction.

Possibly larger doses of toxin might be administered in combination with antitoxin similar to toxin-antitoxin immunization in diphtheria, but preliminary experiments along these lines have not been promising in results. Possibly a method for reducing the toxicity of the toxin, by means of chemical agents, may ultimately prove of practical value.

I fear that toxin immunization against scarlet fever is not yet on a sufficiently sound basis to recommend it as a public health measure on a large scale analogous to active immunization against diphtheria in the schools.

but there can be no doubt as to the advisability of continuing our investigations in order to put it upon a sound basis.

I think antistreptococcus scarlet-fever serum possesses some prophylactic value and especially if there has been intimate exposure requiring immediate protection. One drawback is the high incidence of serum sickness, but this incidence has been materially reduced by the use of concentrated serum.

My experience with serum in treatment of scarlet fever has been quite satisfactory, and I believe that it should be employed in the majority of cases because of its favorable influence in reducing both the mortality and the incidence of complications. My preference is for a serum prepared by the immunization of horses with both the toxin and the streptococci. Possibly the serum could be omitted in the treatment of mild cases, in order to prevent the production of serum sickness; but once in a while a mild case becomes a severe one, and serum sickness is infinitely less dangerous than the disease and its complications.

Dr. Edward L. Bauer: Dr. Park's paper opened with a discussion of diphtheria prevention, and he has offered his usual forceful arguments in favor of the protection of the young child particularly. That is as it should be. His surmise as to our motives for protecting the school child are 100 per cent right. The advertising value of the school child in bringing the lesson of prevention home to parents, the decrease in incidence by having an immune school population, 50 per cent of our cases of diphtheria occur in school children, and by breaking the chain these children will not carry the disease home to the group of greater susceptibles, the infants and preschool-age group, thereby accomplishing the third objective, the lowering a mortality made high by the incidence in this lower-age group. These objectives, we hope, will cut deeply into our diphtheria tables.

The schools will advertise the campaign, but the medical profession must do its share individually and collectively by teaching and practising diphtheria prevention. The work in the schools will be started in West Philadelphia and each school district will follow in order. Dr. Park's claim for a lower incidence and mortality in diphtheria in New York is matched by the claim of McCreary, of the Pennsylvania State Department of Health, that the incidence in Pennsylvania has been cut in half since his active campaign was inaugurated.

Our Division of Child Hygiene has not only coöperated with us but finds that there is an exchange of patients with the baby and runabout clinics that greatly increases the value of all services.

I am not concerned about the possibility of sensitizing children with toxin-antitoxin mixture. I am certain, after my experimental studies recently reported, that this phase is of no importance. Statements to the contrary are not borne out in fact, and are made despite a lack of knowledge of the presence or absence of sensitivity existing prior to the toxin-antitoxin injections.

The subject of scarlet-fever prevention by active immunization is in great turmoil. There is much controversy as to the value of the immunizing agents now employed and great differences of opinion exist as to the value of the antitoxin as a curative agent. We may be running parallel or at a tangent with the truth, but we are not driving with it precisely. There is

some good in it, but as yet it is of little practical value. For example, the smaller doses of Dick toxin give negative Dick tests for a brief time. Following the larger doses there frequently occurs a streptococcic erythema or scarlet fever toxemia, and we do not know just to what extent damage will be done in any given case. Parents properly object to this and more care must be exercised. Numerous strains of streptococci are accredited with the production of scarlet fever. Is it possible to immunize against one strain and neglect the others? My results with the Dick test agree with Dr. Park's. Investigating carefully the histories, especially in institutions where good records are kept, I found that 12 per cent Dick positives occurred in those with *bona fide* histories of scarlet fever. Have they had scarlet fever with one strain and are susceptible to another? Then again, with so high a percentage of susceptibles, why do we not have more secondary cases of scarlet fever? Has Dr. Park used the bactericidal antitoxin, and if so with what result? There are still many other questions still unanswered too, but let this suffice.

So far as the antitoxin is concerned, I have used it and feel that I have gotten results. Years ago, before I began the study of medicine, I recall having heard my father say that following large doses of diphtheria antitoxin quite favorable reactions occured in scarlet-fever cases, particularly in the severer types. The temperature would come down rapidly and the patient recover without complications. This was before the days of concentrated antitoxins. Since the protein content has been reduced I have not heard this claim made. I wonder if the protein in the scarlet-fever antitoxin may not be responsible for the favorable action of this remedy?

To sum up, I must know more about this problem before going to my director and saying, "Here is something we must use in Philadelphia." I am not ready to say that it should be used and I do not agree with Dr. Kolmer that it should be given promiscuously and used in everybody's private practice. That will only permit of accidents that will react deleteriously to the work of such careful investigators as Dr. Park and possibly set their work back generations.

Dr. Park (closing): There were several questions asked by Dr. Bauer and by Dr. Kolmer. One was as to whether the time was ripe to start the routine immunization against scarlet fever? I do not think that we are ready to do it yet. On the other hand, I do believe wherever scarlet fever breaks out in an institution it is very worth while to do it. Also, when persons are exposed in families I personally prefer not to give antitoxin but rather to do a Dick test immediately and give at the same time an immunizing injection of 500 skin-test doses. Each child should be watched and if at any time one shows any sign of developing scarlet fever, it is then given an injection of antitoxin. It is surprising how few contact cases develop scarlet fever even if no preventive treatment is given. It is my experience that this amounts to not more than 10 per cent. The Dick test is read the next morning and nothing further is done to negative children. To Dick-positive children, unless suspicious symptoms of scarlet fever have developed, we give another dose of toxin of 1000 skin-test doses and then in a week 5000 and in another week again 5000. By giving toxin

rather than antitoxin they are freed from the danger of serum sickness. The exceptional child that develops scarlet fever is given a dose of antitoxin. We do not know as yet how long the immunity will last which is produced from the toxin. I doubt if it will last more than one or two years. I have no fear of using the toxin; at the present time we are giving 500, 2000, 5000 and 10,000 skin-test doses to children, and even in those who have a markedly positive Dick reaction, if toxin is properly standardized, we do not get any unfavorable effect from these doses. Occasionally a slight rash appears. The manufacturer necessarily puts more in the vials than is stated on the labels, because he wants the vaccine to have the stated potency for six months. We should know before giving the injections their actual strength.

Let us now turn to diphtheria. As to the necessity of doing a Schick test, it may be of interest to note that in New York we have now for two years been giving as a substitute for this test toxin-antitoxin subcutaneously in more than half of all the children; we have used this method in over 50,000 children and it works really very well as a combined immunizing and Schick-test injection. You give 1 cc. in the anterior part of the arm just above the elbow. The results are read in five or six days and practically every child which shows a positive Schick reaction will show larger reaction from the toxin-antitoxin. It is true there are a few pseudoreactions which confuse us somewhat, but the error is on the safe side. We find it very useful, as it saves an injection and still makes the parents realize that we are saving the children who are immune from unnecessary injections.

I agree with Dr. Kolmer that where you fear or have a considerable amount of reaction that by dividing the amount into 4 or 5 doses you greatly lessen the reactions. I think it is very likely that we will choose the toxoid as the final preparation in place of the toxin-antitoxin. Both give equally good results. The toxoid gives somewhat less reaction in young children, while the toxin-antitoxin gives less in older children. The immunizing results in my experience have been identical. As far as the scarlet-fever toxoid is concerned, I have only used it in a few cases and the immunizing results were not so good as by the unaltered toxin.

In the treatment of scarlet fever with serum we have used either the antitoxic or the doubly potent serum (antitoxic and bactericidal) in a considerable number of cases. In some rather late cases the doubly potent serum did unusually well. For instance, in a child which seemed only moderately sick the serum was not immediately given. Two days after its admission the child grew worse and appeared septic. It was given a dose of doubly potent serum. The next morning the whole aspect of the child had changed, it was no longer septic and made a good recovery. I am not sure enough yet to be willing to state that the doubly potent serum should supplant the antitoxic serum.

As to the importance of the difference in the strains causing scarlet fever, it probably is not a serious factor as the toxins produced by the great majority of strains pretty well overlap each other. Probably 95 per cent of the cases of scarlet fever are due to strains whose toxins are nearly alike. Antiscarlet-fever serum is, I think, just as effective as diphtheria antitoxin. I certainly would not advise scarlet-fever serum in all cases. Those that are mild will get well without any treatment.

FOCAL INFECTION FROM THE LABORATORY STANDPOINT*

By JOHN A. KOLMER, M.D.

Despite the fact that the subject of focal infection has been prominently before the medical profession during the past decade, a good deal of confusion and skepticism of its importance still exists. One ordinarily thinks of only the acute bacterial infections and especially of the specific infectious and contagious diseases in relation to infection, but it has been estimated that 80 to 90 per cent of all deaths are due to some type of infection and the acute specific infections are in reality responsible for but a small part of these. To not a few physicians the subject of focal infection begins and ends with the possibility of a deforming arthritis being related to infected teeth or tonsils, the removal of which may or may not be followed by a magical disappearance of the joint involvements. But the possible importance of simple nonspecific infections of the apices of teeth, the follicles of the tonsils, the accessory nasal sinuses and infection in general of the upper and lower respiratory tracts as well as to a lesser extent of the intestinal and genito-urinary tracts in relation to the infection of more distant tissues, as the synovial linings of joints, the sheaths of nerves and muscles, the eye, heart, kidneys, gall-bladder, biliary ducts and other organs, including even a large number of the psychoses and neuroses, as well as possibly to some of the toxic effects ascribed to the administration of arsphenamin and mercury, and especially exfoliative dermatitis and allied conditions, has been properly emphasized, although conclusive bacteriological evidence of the relationship may be lacking in many instances, our knowledge of the mechanism incomplete and our means of exact diagnosis sometimes quite inadequate.

From the bacteriological and immunological standpoints many interesting and important questions arise which time and space

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do not permit of detailed discussion. What types of infection are especially important as primary foci? What organisms are of special importance in relation to focal infection? Are the secondary foci due to the presence of the organisms themselves or only to their toxins? How are these transported to distant tissues in the production of secondary foci? Does focal infection necessarily imply or demand a specific tissue affinity on the part of the organisms or a peculiar susceptibility of the tissues? Why are some individuals subjected to the secondary lesions of focal infection while others escape with presumably worse primary foci? What can the laboratory do in the way of aiding diagnosis and treatment in a practical and satisfactory manner? These are some of the numerous questions which I shall attempt to discuss in a brief and concise manner.

According to my experience, infections of the apices of teeth, the nasal accessory sinuses, tonsils and bronchi are the most important primary foci in adults. Even in some of those cases of gall-bladder and bile-duct infections believed to be secondary foci, I believe that infections of the upper respiratory tract are of primary importance, followed in sequence by secondary infection of the intestinal tract and of the biliary passages through the swallowing of purulent material, or possibly by direct blood-stream infection of the liver. Likewise in that difficult group of arthritis cases believed to be due to organisms or their toxins absorbed from the intestines, the possibility of primary foci in the upper respiratory tract and mouth first engages our serious attention, although it is apparently possible for some of the bacteria of the normal intestinal flora to produce secondary infections and notably pyelitis by the colon bacillus. The genital organs, and particularly infections of the prostate gland and adnexa, are especially important in relation to the production of gonococcus arthritis, tenosynovitis and endocarditis, although the frequency with which staphylococci, and occasionally even streptococci, are to be found in chronic urethroprostatitis suggests that these organisms, rather than the gonococcus, may be responsible.

But of all the organisms believed to be responsible for focal infection, the group of streptococci are undoubtedly most important, followed in order by the staphylococcus, pneumococcus, gonococcus and colon bacillus. *Streptococcus hemolyticus* and *Staphylococcus pyogenes aureus* have certainly been of primary importance in my experience and especially in relation to focal infections of the teeth,

gums and respiratory tract, particularly of the nasal accessory sinuses. But the possible importance of the organism of influenza, and probably even of the diphtheroid bacilli, in relation to focal infection is to be kept in mind, as well as some of the specific infectious organisms like the tubercle and typhoid bacilli and spirochete of syphilis from the standpoint of establishing primary foci from which distant organs may become secondarily infected.

Whether or not the secondary lesions of focal infection are due to the actual transference of the organisms themselves, or but to their toxins, cannot be definitely answered. It is ordinarily believed that the arthritis and tenosynovitis of focal infection are due to the presence of the organisms, and the frequency with which they are found in blood cultures in endocarditis leads to the same conclusions. But the question has been frequently raised, whether the iritis, optic neuritis and other ocular diseases of focal infection are due to the presence of bacterial toxins absorbed from the primary foci or to the organisms themselves. Altogether too frequently ophthalmologists throw away the bits of iris removed during iridectomy instead of subjecting them to culture and preferably in a medium of harmone glucose broth, but it is my opinion that these and the secondary foci in general are due to the presence of the organisms themselves rather than to their toxins absorbed from the primary focus. But the possibility of bacterial toxic substances alone producing the secondary lesions of focal infection is not to be underestimated and especially in relation to the psychoses and neuroses where, in the opinion of McCarthy,[1] they appear to produce symptoms with greater frequency in individuals with arterial hypotension probably of endocrine unbalance. Furthermore, the soluble proteins of bacteria absorbed from primary foci may be responsible in part for allergic sensitization, especially in relation to bronchial asthma, nervous rhinitis and some of the skin diseases, particularly exfoliative dermatitis following the administration of arsphenamin and its congeners.

The distribution of organisms and their toxins would appear to be largely by the blood. Absorption into the lymph draining the primary focus probably is of first occurrence and alone responsible for secondary foci situated in the direct line of drainage, but distribution to more distant organs is almost certainly by way of the blood stream. In this manner the question of the bactericidal and antitoxic qualities of the blood may be important factors in deter-

mining whether or not secondary infections occur, as in all probability these are prevented in many instances by a destruction of bacteria or a neutralization of their toxic products by the blood, even though these immunological agents may be ineffective in the sterilization of the primary foci.

But in case the blood is unable to destroy organisms finding their way into it, what governs their subsequent localization in distant organs and tissues? Does this necessarily demand that the organism shall acquire in the primary focus a specific elective affinity for a particular organ or tissue? Or does it mean that the organism or its products circulating in the blood meet with widespread immunological resistance in all but those tissues or organs of secondary localization? Or is localization to be explained on anatomical grounds in which the process is favored by sites of reduced resistance due to trauma in a broad sense, disturbances of circulation with closed capillary areas and embolism? In my opinion the importance of acquired selective tissue affinity on the part of the organism has been greatly overemphasized, as I believe is true of the so-called specific neurotropic and dermotropic strains of *Spirochæta pallida* in relation to localizations in syphilis secondary to the primary lesion or chancre. Indeed the theory of acquired elective affinity on the part of bacteria in relation to focal infection and the difficulty or impossibility of its demonstration by animal experiments may be one factor responsible for the skepticism of physicians regarding the importance of focal infection in the production of disease, and in my opinion it is not at all necessary to believe in this theory in relation to the subject. I am particularly impressed with the importance of general virulence of the organism, local tissue immunity, hereditary influences and trauma in a broad sense as the determining factors, although space does not permit here a detailed discussion of them.

But in my opinion these agencies also largely determine why some individuals are more prone to secondary focal infections than others. The individual showing the largest shadows or areas of cavitation and radiolucence about the apices of teeth are not always the ones showing secondary localizations or lesions of focal infection. Indeed, as stated by Price,[2] the occurrence of these areas of rarefaction may be interpreted as evidence of a good local reaction and a high immunological defense against systemic infection, with well-marked bactericidal activity of the blood sufficient for protecting against

secondary infections until these immunological agencies are broken down by some kind of systemic overload, among which intercurrent acute infection, anemia, undernutrition, hypotension, endocrine unbalance and a reduction of ionic blood calcium may be of importance.

Unfortunately organisms may survive in the blood long enough for their transference to distant tissues and organs even though they may not be able to multiply in the blood and thereby produce the clinical evidences of bacteriemia or septicemia. But the matter of secondary localizations or lesions in focal infection involves not only the virulence and numbers of organisms gaining access to the circulating blood, but likewise the kind and degree of local resistance about the primary focus or foci, the degree of systemic humoral and cellular resistance and the question of predisposition to secondary infection by anatomical defects and the presence of areas of inherited or acquired reduced tissue resistance.

Finally, what may the laboratory do in aiding the diagnosis of focal infection? Extracted teeth may be reliably cultured, as likewise infected sinuses, tonsils, the secretions of the respiratory tract, prostatic secretions, etc. The finding of streptococci, especially the hemolytic and viridans types, is always of considerable significance, as likewise the presence of *Staphylococcus aureus* and the *pneumococcus*. But the laboratory can do but little beyond these cultural examinations, and bacteriological studies of the secondary foci like those in the iris and other parts of the eye, the joints and sheaths of muscles and nerves, etc., are usually impossible, although far more important in clinching a diagnosis of focal infection than an investigation of the primary foci. The one exception is the detection of bacteria in the blood in septic endocarditis and especially of streptococci, although even this may require several attempts and careful attention to technic before positive results are obtained. Certainly merely finding streptococci, staphylococci or pneumococci in the crypts of the tonsils or secretions of the respiratory tract does not establish the existence of focal infection and the same may be said of bacteriological examinations of the feces, but the finding of these and other organisms in cultures of closed spaces like apical abscesses, the sinuses, and gall-bladder are of much greater significance, because by proper attention to technic and detail secondary contaminations may be reduced to a minimum or entirely excluded.

But even when organisms are recovered from primary foci

under acceptable conditions, can their relationship to secondary focal lesions be proven? In the first place, the instrumentation necessary, as the extraction of a tooth, enucleation of a tonsil, curettage of a sinus or administration of an autogenous vaccine, may be followed by an exacerbation of the secondary foci and such are always strong presumptive evidence of a relationship between the foci of primary and secondary infection. Determining the virulence of the organism for the rabbit or other lower animal may be of aid, as likewise the question of elective localization of the organism in the corresponding organs of this or some other suitable animal possessing a reasonable degree of susceptibility. But the absence of virulence and the absence of evidence of elective localization of the bacterium in the healthy lower animal does not exclude the possibility of its being responsible for focal infection in the sick human being. On the other hand, the demonstration of virulence and elective localization is strong presumptive evidence in favor of regarding the organism as the etiological agent, although even here the matter of localization in the lower animal need not always involve the question of acquired selective tissue affinity on the part of the bacterium.

Is a measure of the immunological resistance of value? Unfortunately a determination of the bactericidal activity of the blood is not always to be accepted because, in my opinion, the blood of a patient capable of preventing the growth of bacteria in the test-tube may not prevent the establishment of primary foci of infection, nor kill off the organisms during the intermittent and probably fleeting periods of bacteriemia during which the organisms are being transported to distant tissues and organs. But a bactericidal blood-test along the lines developed by Cohen and his associates is of value in estimating the chances of a generalized blood infection from a primary focus even though of limited value in relation to focal infection. In other words, it would appear that when the blood is bactericidal or bacteriostatic *in vitro* for an organism the latter is incapable of producing a true septicemia, the blood may be unable to sterilize a localized focus of infection or to kill those bacteria gaining access to it for brief periods of time. At present there is some hope for developing a successful complement-fixation test in the diagnosis of focal infection and such would prove a valuable and welcome addition to our methods of diagnosis.

In conclusion, the laboratory may aid in the treatment of focal

infection by the careful preparation and administration of autogenous bacterial vaccines. If one could be sure that the secondary foci are maintained by constant infection from the primary foci, the removal of the latter would be all that is essentially required in treatment; but unfortunately the infection may be so well established in the secondary foci that removal or drainage of the primary lesions has little or no direct influence upon recovery. For this reason drainage or extirpation of the secondary foci may be of equal importance; but when this cannot be done I believe that it is sound practice to include the careful administration of a properly prepared autogenous vaccine as part of the treatment in an attempt to aid in the walling off or eradication of the secondary bacterial deposits. Even in cases without clinically detectable secondary foci, especially those of apical abscesses and chronic sinusitis, I believe it is sometimes wise and well to include the administration of four to six doses of autogenous vaccine for prophylactic purposes in aiding the eradication of clinically latent or undetectable secondary lesions which may be present.

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1. Jour. Am. Med. Assn., 1926, lxxxv, 1949.
2. Jour. Am. Med. Assn., 1925, lxxxiv, 254.

FOCAL INFECTION FROM THE VIEWPOINT OF THE INTERNIST*

By O. H. PERRY PEPPER, M.D.

The internist finds himself between the horns of a dilemma in this matter of focal infection. On the one side is the undoubted fact that an occasional case obtains almost miraculous relief as a result of the discovery and appropriate treatment of a focus of infection. On the other side, he is faced by his realization that we are today at the height of a focal infection fad, which we are probably carrying much too far and in the pursuit of which we are sacrificing enormous numbers of teeth and tonsils and carrying out expensive and sometimes dangerous procedures against foci of infection, the harmfulness of which we are unable to estimate.

It is only some fifteen years ago that the term focal infection first appears in the index of the *Journal of the American Medical Association*. Of course, that was not the beginning of the interest in this subject, but previous writers, and there were not many of them, had for the most part dealt with the relation of some one focus to some one systemic or metastatic lesion. Within the past fifteen years we have seen the literature on this topic grow to an enormous degree. Each year lately the *Cumulative Index* has had a page or more of references concerning focal infection, and yet today we must admit that there are many questions concerning this subject which we cannot answer and that there are many arguments which can be advanced against it and which we cannot refute except by quoting clinical results.

It is only twenty-five years since we saw the rise and fall of interest in the subject of movable kidney and its systemic effects. It is instructive and humiliating to go back into the literature of twenty-five years ago and read the many articles describing the variety

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of conditions and symptoms attributed to the movable kidney and the remarkable therapeutic results obtained by nephropexy. Among the symptoms accepted as due to movable kidney were nervousness, fatiguability, headaches, depression, insomnia, cardiac palpitation, gastric indigestion, constipation and vomiting. The literature of that day was just as sincere and just as conservative as is the literature of today. They were just as sure of the efficacy of nephropexy as were the writers of fifty years ago of the value of removal of the ovaries and the writers of today of the removal of foci of infection. Remarkable clinical cures were obtained by nephropexy of conditions for which that procedure is not thought of today. Today no one would deny the many remarkable cures obtained by tonsillectomy and extraction of teeth with abscessed roots. Are we merely in the midst of a movement which will run its course and be looked back upon in ten years as we look back on the days of nephropexy? Or are we still merely on the edge of a proper appreciation of the significance and importance of focal infection? Unquestionably the judgment of those in the midst of such a movement is poor. We may not be capable of deciding for ourselves, but at least we may ask ourselves the question and appreciate the possibility of our error. Furthermore, we can frankly admit our lack of knowledge of some of the essential steps in the argument and theory by which the action of focal infection is explained.

We have no method of telling whether a given focus of infection is causing systemic symptoms or not. Of course, the therapeutic test may in certain instances answer this query. But upon the discovery of a sinusitis, a chronic prostatitis or an abscessed tooth root, we cannot say whether this focus is causing the patient's arthritis, iritis or neuritis. A means of answering this question is very much needed. The bacteriological difficulties of this problem have been detailed to you. The streptococci still offer great difficulties to the investigator. A careful repetition of Burbank's work is needed and may perhaps give a lead, but until confirmed this method must be accepted with caution. Similarly, the claim that the pathogenic nature of a focal infection can be revealed by a local flare-up following a nonspecific protein injection needs further investigation.

Unless we take the position that every focus is guilty, we must often be in doubt as to the advisability, or at least the necessity, of radical removal of various foci. There will be little doubt in

the case of a patient whose sight is threatened or whose livelihood is endangered. In such a predicament any of us would sacrifice teeth and tonsils and submit to sinus drainage and prostatic massage. But we must feel somewhat less certain as to the need of eradication of every focus in a patient with some condition not commonly included in the list of those caused by focal infection, if there are any such conditions left. For example, how far should one go in the treatment of foci of infection in a nervous, apprehensive patient with exophthalmic goiter? How thoroughly convinced of its harmfulness would you have to be to permit the removal of your colon as a focus of infection?

Still greater doubt must exist when dealing with the supposedly healthy individual. If we are to do our duty by those who come to us for annual examinations, we must be prepared to give an opinion as to the necessity for the treatment of symptomless foci of infection in the otherwise healthy individual. One may say that it is the symptomless foci which are the most dangerous and that the degenerative changes which come with age are in part at least the result of chronic intoxication from focal infection. Others may not admit this view. Certainly it is not proved.

Another question which we are unable to answer satisfactorily concerns the interpretation of the reaction which may follow the eradication of a focus of infection. Is an exacerbation of the metastatic lesion an argument in favor of a causal relation between the eradicated focus and the metastatic lesion or not? Is such a reaction specific or may it be nonspecific? If benefit follows the eradication of a focus do we know that we have done anything more than treat our patient with nonspecific protein shock?

What are the possible sites of focal infection? All would agree on the sinuses, the middle ear, the tonsils, the prostate and peridental infection. But the enthusiasts would have us include the gall-bladder, the appendix, the cervical and peribronchial lymph nodes, the large bowel, the rectum and under certain conditions the skin. Each of these deserves full discussion if time but permitted. Each presents special problems.

Tonsils and teeth are today the sites most suspected and one cannot help wondering whether the fact that they are the most easily removed foci does not subconsciously enter into this tendency. Undoubtedly the removal of tonsils has gone too far. In my experience it is unusual for the removal of tonsils to be effective in the

relief of a metastatic lesion unless there have been recurring attacks of acute tonsillitis or definitely enlarged peritonsillar lymph nodes. Peridental infection is far more important and the more youthful the individual the more important peridental infection is. Unfortunately the peridental lesions least evident in the roentgenogram are the very ones which give profuse cultures of streptococci, while the larger obvious abscesses are very apt to have become sterile.

Mild, symptomless sinusitis is so common that we must expect it to be found in a percentage of all patients examined and we must not be too quick to accept the discovery of ethmoiditis, for example, as the sure explanation of some metastatic lesion. Sinusitis is probably the most frequent and important focus of infection in children and deserves much of the attention now devoted to the tonsils. Nor should the middle ear and mastoid cells be forgotten. I have seen a residual mastoid infection explain a persistent hemorrhagic nephritis in a child.

Nonvenereal prostatic infection deserves the emphasis which it has received from Neilson,[1] of St. Louis, Irons[2] and myself.[3] The most spectacular cases of relief of metastatic eye lesions by the eradication of foci of infection have, in my series of cases, followed treatment of nonvenereal prostatitis. These cases show streptococci in the prostatic secretion and are, I believe, instances of blood-borne infection initiated from a preceding tonsillitis.

The evidence justifying the inclusion of the gall-bladder, the appendix, the cervical and peribronchial lymph nodes, the apparently intact mucosa of the pharynx, bowel or rectum in the list of foci of infection is less certain, although Irons[4] lends the weight of his approval to most of these foci. Of course, any ulcerated surface may offer a portal of entry to a variety of organisms which perhaps had nothing to do with the production of the ulcerated lesion. Such lesions may occur in the bowel, as for example in amebic dysentery, or in the nasopharynx. The invading organism may set up a metastatic lesion at a distance.

Unfortunately, also, an individual with one focus of infection seems always to have more than one; when one finds one peridental abscess there are usually others. It is as though there were some

[1] Med. Clin. North America, 1924, vii, 1669.
[2] Jour. Am. Med. Assn., 1923, lxxxi, 1770.
[3] Illinois Med. Jour., 1924, xlv, 93.
[4] Canadian Med. Assn. Jour., 1924, xiv, 205.

basic general reason, some anatomical or immunological lack of resistance. Where more than one focus is discovered, treatment becomes proportionately more uncertain and difficult. Treatment should not be begun until by thorough study one has discovered all the foci which can be demonstrated. Then only can a proper program of procedure be decided upon.

In this connection it is important to realize that there exists confusion between a focus of infection and a portal of entry. These two terms mean different things and there is a fundamental distinction between the two. The fact that the tonsil acts as a portal of entry for the streptococcus which lodges on the damaged heart valve of rheumatic carditis need not constitute the tonsil a focus of infection. On the other hand, the acceptance of a sinus as a focus of infection in a patient with neuritis does not in any sense prove that it is or has been a portal of entry. This distinction is important, for the indications for the radical treatment of a portal of entry may be very different from those in the case of a focus of infection.

The same confusion exists as to what conditions may result from focal infection. The clearest examples occur in the ophthalmological field. In the sphere of internal medicine neuritis, arthritis, purpura and nephritis come first to mind, and clinical results seem fully to justify the opinion that each of these conditions may be abruptly relieved or improved by the eradication of a focus of infection. Less sure are we as to gastric and duodenal ulcer, although some of the best of the experimental evidence supports this possibility. Hyperthyroidism has been much discussed in this relation, but proof is wanting. Even more difficult to judge is the question as to a focal-infection basis for myocarditis, cholecystitis, herpes zoster, eczema and secondary anemia, and yet each of these is unhesitatingly accepted as often due to focal infection by the ardent advocates of this theory.

Also the theory has been applied to explain insanity, neurasthenia and the various psychoses. It is in this field that the most radical application of the focal infection theory has been carried out. It is true that patients with mental disease are a group in which one is perhaps justified in taking radical measures in the hope of benefit even to a small percentage, but when in addition to the eradication of all suspicious teeth and tonsils, the operations of removal of the colon and amputation of the cervix of the uterus are performed, for

the purpose of removing foci of infection, it makes one doubt. In 250 colectomy operations thus performed for the relief of psychoses the death-rate was 30 per cent. Hobbs[1] made a survey of psychiatric opinion as to focal infection as causative factors in functional psychoses and found that the opinion was almost universally opposed to the measures taken and the claims made by the more radical of the enthusiasts in this field.

It is radicalism such as this which characterizes all fads at their height and helps, by bringing discredit upon the procedure, to start the swing back to a saner level.

Back of our difficulties in forming a judgment of this focal-infection theory is our lack of an acceptable and experimentally demonstrated explanation of the action of focal infection. Until that is available we must rest our belief in the theory upon clinical evidence, and this foundation, we know to our sorrow, must be built slowly and with the greatest caution.

The theory has been advanced that the organisms concerned in chronic foci of infection each exhibits a remarkable and specific tendency to produce metastatic lesions only in some one certain tissue of the body. This theory has not been unanimously accepted by bacteriologists and experimentalists, but has received support from several quarters.[2,3,4]

It is a temptation to the physician to accept this as a basis for the focal-infection theory, for, if true, it supplies us with a much-needed explanation and justification. We must have a satisfactory scientific basis to justify a full adherence to the focal infection theory. At present we try to effect a compromise. Our activity in the treatment of possible foci of infection depends upon the severity and nature of the metastatic lesion, on the one hand, and on the other, on the danger of the treatment of the focus. This is unsatisfactory.

Until we get such an explanation we must carefully and conservatively apply our best clinical judgment both in the decision as to the eradication of each focus of infection in each individual case and even more so in the interpretation of the results of such therapy.

[1] Jour. Ment. Sci., 1924, lxx, 542.
[2] Arch. Int. Med., 1923, xxxii, 828.
[3] Am. Jour. Med. Sci., 1925, clxix, 407.
[4] Ann. Surg., 1924, lxxiv, 29.

FOCAL INFECTIONS AND OCULAR DISORDERS*

By G. E. De SCHWEINITZ, M.D., Sc.D.

Did time permit it would be interesting to review the history of the so-called doctrine of focal infections, because observations along these lines are of fairly ancient lineage. At least it may be permitted to remind the Fellows of the College that Benjamin Rush more than one hundred and fifty years ago described the cure of disease by the extraction of decayed teeth.

Coming at once to out own period, it is safe to assert that in our country the development of the doctrine of focal infection "from bacteria of the streptococcus-pneumococcus type *via* the teeth, tonsils and other portals" (Garrison), dates from about 1909, and is largely due to Frank Billings and E. C. Rosenow.

Although it is well-nigh fifty years since Nettleship, in England, noted the etiologic relationship of a carious tooth to choroiditis with keratitis punctata, it is especially within the last sixteen years that English ophthalmologists have concerned themselves with the study of local infections, notably in the teeth, and ocular disorders. During the same period an increasing interest in this matter has obtained in our country, and this relationship continues to be widened, and not only the choroid, ciliary body and iris inflammations are credited to a metastasis from local foci of infection, but also certain affections on the cornea, sclera, retina and optic nerve. Indeed, at the present time no conscientious ophthalmologist would presume to treat such eye disorders without an investigation of those areas which are commonly regarded as likely to harbor infection.

In May, 1924, in Paris, La Société française d'Ophtalmologie devoted one session to focal infections, especially in the teeth, and ocular disorders, and Dr. Camille Fromaget presented an admirable,

* Read May 5, 1926.

comprehensive review of this whole matter. On that occasion I had the privilege of delivering a lecture on some types of metastatic (focal) infections of the eyes, and prefaced this essay by a restatement of certain principles which pertain to focal infections in general, but also with particular emphasis on ocular disorders. This paper has been published only in the *Bulletin* of the Society, and I permit myself to quote freely from it, with some modifications and additions, now, of course, with reference to the eye.

Metastatic and often recurrent infections (inflammations) of the eye may result from distant chronic local infections, and their removal (*i. e.*, the focal infections) is frequently, but not invariably, followed by disappearance of the metastatic lesions, and of the recurrences.

It is assumed that bacteria cause the primary lesions, although it has not been possible thus far to demonstrate a complete mechanism for each organism. E. V. L. Brown has succinctly thus stated the facts in question: Bacteriemia has necessarily been only assumed in many cases, and yet bacteria should be demonstrable in the blood stream if the evidence in the case shall be unquestioned. This unassailable evidence is difficult, perhaps impossible, to obtain, because only a few organisms are present in the blood at a given time; if it were otherwise a condition of sepsis would develop.

Animal experimentation has demonstrated that cultures from an infected area (focal infection) injected intravenously may produce a metastatic inflammation in an eye, a culture from which in its turn injected into the veins of other animals will create an identical lesion, the evidence being especially clear in relation to abscessed teeth and iritis (Rosenow, Benedict, E. V. L. Brown, Irons, Park Lewis, Haden, and others). But naturally, the entrance of microorganisms into the blood streams does not imply that metastatic lesions must arise in the eye, because the bacteria may be destroyed in the blood stream, but only that they may become active in the production of such lesions if resistance to infection is depressed.

The selective tissue affinity of certain bacteria, *i. e.*, elective localization, is a theory which rests, in the opinion of some expert bacteriologists, upon a satisfactory foundation, and it is believed that the bacteria may locate in certain ocular tissues, either because they have developed a special affinity for these parts and find conditions favorable for their growth, or because the resistance of these parts is reduced by some other agency and thereby favors the localization of the germ.

The agency which reduces resistance in these circumstances may be regarded as an injury which puts out of operation local protective forces, and this injury may be chemical, physical or depend upon a chronic infection. The subject of a remnant of gonorrhea in the posterior urethra (it may be years after the incidence) acquires an iridocyclitis after excessive reading. This is an example of a physical injury, which neutralizes the protective forces of the part involved. A person with chronic syphilis or other chronic infection may beget, for instance, a uveitis, which, resisting the usual remedies, yields to them after the elimination of a focal infection (an abscessed tooth or septic tonsil, etc.). This is an example of the coöperative effect of a luetic or other chronic malady and secondary (pyogenic) infection. Or gout, diabetes or hypothyroidism and an area of chronic (focal) infection may coexist. An ocular inflammation ensues because the disordered metabolic products furnish a chemical injury which reduces the resistance in an area against which the bacteria from a focal infection launch a successful attack. These effects of associated influences are in part recorded as the result of consultation with Dr. Flexner.

I am aware that the theory of E. C. Rosenow that bacteria have a tendency to lodge themselves in certain tissues of the eyes, for example, for which they possess specific combining affinities, is not accepted by many bacteriologists. R. L. Haden, however, whose experimental work on infected teeth in relation to iritis appears to confirm Rosenow's contention, believes that those who have failed to verify Rosenow's results have also failed in most instances to carry out with care his technic.

Be that as it may, bacteria coming from the primary lesion (focal infection), which is suppurative, may cause a nonsuppurative metastatic ocular inflammation, because in their contact with the blood serum their virulence is decreased; they may be found in the environment of the affected lesion (for example, staphylococcus, etc., in the aqueous humor in focal iritis), or in the substance of inflamed tissue (for instance, streptococci in the iris (Rosenow, Brown, Irons, Nadler)), or they may be absent, having been destroyed by the virulence of the inflammation which they have produced. Whether these forms of metastatic inflammation are ever produced by circulating bacterial toxins instead of the bacterial element itself is still in controversy. Although no one has proved that circulating bacterial toxins do not exist, which may find tissue elements for which they possess a specific combining affinity, it is

highly probable, in most circumstances, that in bacterial infections the terms themselves are present rather than their products as toxins and endotoxins produced in a distant focus.

The elimination of one focus of infection does not imply that the source of the metastatic inflammation has been removed; the foci may be multiple. A recurrence of the ocular inflammation may be due to a bacterial invasion from an area of infection not previously discovered.

Brown Pusey offers the following explanation of a recurrence of the inflammation: "In the first attack a depot of bacterial emboli is established in the iris, for example. The reaction to this metastasis completes its course, that is, it disappears, but leaves the area sensitized to the protein of the embolic bacteria. A later infection occurs (the place is of small importance) caused by this strain of bacteria, whose specific protein is thus introduced into the organism, where, being absorbed, it reproduces the inflammation in the sensitized zone."

After removal of a focal infection, the improvement in the metastatic ocular infection, presumably due to it, may be *slowly* progressive, or it may take place *rapidly*, and in a few hours the whole process may disappear, moreover, permanently. Such rapid results are probably similar to those which occasionally follow intravenous injections of foreign proteins, and, like them, are probably nonspecific (Irons).

Many varieties of pathogenic bacteria have been accused with respect to the ocular effects of focal infection—the *Staphylococcus aureus and albus*, *Micrococcus catarrhalis*, the *Streptococcus hemolyticus*, *viridans* and other strains, the *Pneumococcus*, the *Colon bacillus*, *Bacillus pyocyaneus*, *tubercle bacillus*, etc.

It is possible that future investigations shall establish a definite relationship of certain bacteria, or bacterial strains, to definite lesions for instance, the *Streptococcus viridans* of dental sepsis to one variety of focal iritis.

Local or focal infections in their relation to ocular disorders may be found in various structures of the body, common situations being in the teeth, the tonsils, the paranasal sinuses, the intestines, the posterior urethra and prostate, the seminal vesicles, the bladder, and the pelvic organs in woman; less common ones are the upper respiratory tract, the tissues in the hila of the lungs, the gall-bladder, the appendix and the skin (boils, etc.), the nasal mucous membrane,

etc., but it is impossible to define with exactness the favorite port from which the invading bacteria enter the blood stream. In the tabulation of E. V. L. Brown and E. E. Irons the tonsil is accorded the first place in 200 most carefully investigated cases of iritis, or, more exactly, 26 cases were attributed to tonsil infection alone, but only 12 cases to dental infection alone. But this simply proves, as the authors themselves observe, that in this particular series tonsillar infection predominated. Dental infections might readily exceed those from the tonsil in another series. Thus, in an analysis of 100 consecutive cases of endogenous (focal) iritis by Albert E. Bulson, Jr., of dental infection, there were approximately $33\frac{1}{3}$ per cent; of tonsillar infections about 20 per cent.

Again, there may be two or more possible ports of entry, for example, tonsils and teeth. Irons and Brown in 24 cases of iritis where there were multiple or combined infections, were unable to determine which one had given rise to the iris inflammation.

It is probable, as these authors contend, that multiple infections "have a cumulative effect in reducing the resistance of the patient," and in these circumstances it is difficult, if not impossible, to decide which infection is responsible for the ocular inflammation. They further maintain, and rightfully, that the elimination of one infection may alter conditions so that the patient is enabled to recover from the effects of the others. It is also probably true, as Rosenow states, that eradication of infected teeth, for example, may be efficient in dissipating an infection of the tonsils, which was previously coöperatively active. It is evident, therefore, that "conclusions based on the results of treatment must be guarded" (Irons and Brown).

But it is also true that therapeutic success is often not attained until each and every area which may harbor local sepsis "is cleaned up," to use a common expression.

In certain circumstances a chronic local infection (focal infection) may be responsible not exactly for a metastatic inflammation, as it is ordinarily understood, but for a disorder (endovasculitis, sclerosis) of the small vessel supply of a defined area of tissue in the retina or choroid, which in turn determines degeneration of the part thus deprived of its nutritive supply. It is difficult to escape the conviction that in these circumstances the circulation of toxins may be active.

Evidently, in summarized repetition, the removal of one area

of focal infection, followed by improvement in, or cure of, a distant tissue lesion, does not prove that chronic local infection was the source of the bacteria or toxin which caused the metastasis; it may have been due to an undiscovered focus, or to another factor.

Although almost none of the ocular tissues possesses an immunity from the toxic elements which proceed from distant areas of focal infection, it is true that the structure of the uveal tract (iris, ciliary body and choroid) is more frequently attacked than the other parts of the eye, but it would be ill-advised not to emphasize that "these other parts" (cornea, sclera, retina and optic nerve) may also be involved, sometimes in association with inflammation of this uveal tract, that is, indirectly, but also without such association, that is, directly.

As already stated, there are a great many regions in the body which may harbor focal infection, but in practical work the teeth, tonsils, paranasal sinuses and intestinal tract are most frequently accused.

I am unprepared to say that G. K. Swinburne is correct in his declaration that nonvenereal infections of the prostate gland should rank with chronic tonsillitis as a depot of focal infection. But I am sure that chronic nonspecific prostatitis and bladder infections entirely deserve the increased study they are now receiving in the relationships at present under discussion.

As to the sexes, there appears to be no difference of importance in the incidence of autotoxemic iritis and iridocyclitis. As to the age of patients, this may be said, that they (iritis and iridocyclitis) are comparatively rare in children, very rare prior to puberty.

Among the more uncommon ocular morbid manifestations attributed to focal infections are: (1) Areas of central retinochoroiditis, hemorrhagic and otherwise, even those which are so common in elderly subjects. In these circumstances regions which should be under suspicion are the prostate, the bladder the tonsils and the teeth, including the edentate gums which may harbor infection as potent as that which exists in septic teeth. (2) Nonprominent infective optic neuritis and retrobulbar (axial) neuritis. Naturally, the paranasal sinuses most often are responsible for such infections, but the teeth, tonsils and intestinal tract must not be disregarded, nor other situations less frequently guilty.

Evidence accumulates, both clinical and chemical, that a relationship exists between intestinal sepsis, in pockets or generalized, and the development of certain types of toxic amblyopia, notably

those ascribed solely to tobacco and alcohol. The influence is similar to that of diabetes, which, as is well known, renders the optic nerves of its subject peculiarly liable to the poison of tobacco.

Various types of episcleritis are undoubtedly related to focal infection, and so are some forms of ulcerative and other manifestations of keratitis The paranasal sinuses and the nasopharyngeal areas, as was long ago noted, deserve in this relationship particular attention; also the bladder, as there appears to be a form of inflammation of the cornea directly due to colon bacillus infection proceeding from this viscus.

There is some evidence that local (focal) infection is a possible etiological factor in multiple sclerosis, ranking in this respect with the significance of various general infections, as originally suggested by Pierre Marie.

Sinus infection (ethmoiditis and sphenoiditis) appears to be the most important focus when the first stage of multiple sclerosis manifests itself as an axial optic neuritis (retrobulbar neuritis), as Dr. E. A. Shumway has pointed out, but infected tonsils and teeth cannot be disregarded.

Although no positive proof has been uncovered that a focal, usually sinus, infection is more than "a predisposing influence for the true cause or an aggravator of a disease already started by the true cause" (Barker), observations are not wanting which indicate that a relation exists which is more intimate than the sentence quoted implies.

It is possible that future investigations may demonstrate a specificity of the bacterial elements in those sinus infections which originate an axial neuritis as the primary manifestations of multiple sclerosis, exactly as recent observations indicate that there is a specificity of certain microörganisms in the etiology of other diseases of the nervous system.

While it is generally believed, and properly, I think, that an etiological relationship definitely exists between septic foci in various areas of the body and certain diseases of the eye, notably of the uveal tract; while it is commonly accepted that the removal of the focal infections which may be responsible for metastatic ocular inflammations is the proper procedure; and while autogenous vaccin therapy has proved its value under certain conditions, there is room, plenty of it, for additional studies, and it is clearly necessary to exercise balanced judgment, and avoid, shall we say, indiscriminate accusations of focal infections.

SURGICAL ASPECTS OF FOCAL INFECTION*

By GEORGE P. MULLER, M.D.

The subject assigned to me is too comprehensive to be covered in a brief time and I doubt if I could do full justice to it anyway. We must begin with the premise that the streptococcus is the organism most prominently concerned, and that the mouth—especially the teeth, tonsils and the nasal sinuses—are the usual portals of entry. I wonder whether most nose and throat surgeons will go as far as Gluck (1925) when he says "that the tonsils cannot, under our present community life (in our present state of civilization), remain normal. In every individual case they must invariably become and remain infected." There are many statistics on this point. For instance, Blenton (1919) asserts that the streptococcus could be obtained from the tonsils of normal persons in 90 per cent and from the depths of the tonsils in 80 per cent; Pilor and Davis (1919) found hemolytic streptococci in the crypts of the faucial tonsils in nearly 100 per cent. There is no doubt that appendicitis, cholecystitis, gastric and duodenal ulcer and very probably nephrolithiasis and ulcerative colitis are brought about by microörganisms, particularly the streptococcus, from a focus in the teeth and throat. Surgeons have noted the almost epidemic-like frequency of appendicitis following in the wake of the streptococcus throat colds, and one of our Fellows, J. S. Evans, called attention to this in 1918.

The researches of Rosenow have become classic. Beginning in 1913, he showed the elective localization of the streptococcus in gastric ulcer and later extended this to include cholecystitis, appendicitis and pancreatitis. The normal intact mucous membrane of the gastrointestinal tract affords marked protection against the invasion of the streptococcus. This is well shown in the paper by Davis (1920), who in 53 cases was unable to find hemolytic strepto-

* Read May 5, 1926.

cocci in the feces in any case; he believes the protection lies in the gastric secretion, hence the extreme importance of the proper acidity of the stomach. High acidity is constant in peptic ulcer, and hence, if Rosenow's work is confirmed, streptococcus infection through the blood stream and usually from infected teeth or tonsils, is of high importance. In the case of the gall-bladder, Rosenow (1921) found that the most marked lesions occurred when a *mixture* of the streptococcus and colon bacillus was injected. The association of cholecystitis and ulcer is frequently noted by surgeons, and last November, in the series of chronic gall-bladder disease which I reported before this College, 6.5 per cent had both conditions. Rosenow found that when he injected ulcer strains 21 per cent of the animals developed gall-bladder lesions, and when he injected gall-bladder strains 29 per cent showed ulcer lesions. It may be that the same will be found to hold true in the case of appendicitis and that the appendix is not the frequent cause, *per se*, of gall-bladder disease or ulcer, but rather is susceptible to similar strains of bacteria. On the other hand, we may believe that the appendix, for mechanical reasons, acts as a test-tube with permeable walls, and the continued growth and regrowth of organisms furnishes a focus of infection which ultimately reaches the liver and either through the bile or by way of the lymphatics passes into the gall-bladder. The gall-bladder then may act as the focus, and either send the infection to more distant tissues, such as the joints, or through the lymphatics to the pancreas. The following case is of interest in this connection:

Male, aged forty-eight years, suffered since 1903 from attacks of rheumatism, especially in the knees and ankles. In 1916 tonsillectomy was done and he was relieved for a short time. In December, 1924, he was markedly incapacitated and examination revealed disease of the teeth, a number of which were extracted with considerable improvement, although the right wrist and hip were painful, swollen and crippled. Early in 1925 he became worse and came under my observation on March 19, 1925, suffering from a recent attack of cholecystitis. The gall-bladder was enlarged and the *x*-ray showed the presence of a stone. On March 20, a cholecystectomy was done and one stone found imbedded in the cystic duct. The appendix was much swollen and almost acutely inflamed. It was removed. Forty-eight hours later the improvement in the joints was distinctly manifest, and although the patient suffered a very stormy convalescence, he ultimately recovered, continued to improve, and at the present time, one year after operation, is apparently perfectly well. He weighs 200 pounds and works daily.

Another phase of gall-bladder infection which leads us to believe that this organ acts as a focus, is the effect of cholecystectomy, or even simple drainage, upon certain cardiac conditions. It has long been known among physicians, as well as surgeons, that a chronic myocarditis occasionally accompanies gall-bladder disease and especially if there are gall-stones. The influence of chronic vagal irritation must be remembered, but it seems likely that there is also some other influence in the form of a selective toxemia. Moynihan speaks of these as "cholecystic hearts," and in latest edition of his *Operative Surgery* states that he has seen many cures following cholecystectomy.

The importance of treatment of an infected gall-bladder is also apparent from some remarks in a paper by Judd (1921). In series of associated hepatitis with gall-bladder disease, a group was followed up in which the changes in the liver had advanced to cirrhosis. In spite of the diseased liver, 17 of 18 patients traced report that they have been practically well since the operation for cholecystitis.

If we turn to the kidney we find the evidence somewhat conflicting. Perhaps the preponderance of evidence favors the belief that kidney stone is a problem in colloid chemistry, and that infection plays its chief rôle in producing suppurative processes. The acute suppurative hematogenous nephritis is a conspicuous example and is usually caused by *Staphylococcus aureus*. Much could be said about the prostate and seminal vesicles. These organs may be infected from a focus in the teeth, tonsils or gastrointestinal tract, and then may in turn act as the focus. Many brilliant cures have followed prostatic massage, and I believe that in most joint affections the prostate should be investigated with the same care as the tonsils or teeth. In severe cases good results have been obtained by prostatectomy and removal of the seminal vesicles.

Another focus of infection rarely thought of except as a local lesion is endocervicitis. Not only may the infection of the glands under the mucosa produce a chronic cellulitis of the pelvic tissues, and consequent backache, but the infecting organisms, especially if they are streptococci, may cause joint lesions and myocardial or renal affections.

The infections so far described are largely chronic in nature, but surgeons often see serious results from a primary focus. An interesting example occurred in my practice some years ago. The patient was a young boy who was hurt in some way and sustained

a lacerated wound of the left forehead and a bruise of the right thigh. The skin of the thigh was not abraded. The wound was sutured in a hospital and the boy sent home. In three days his family physician reopened the wound because of infection. A few days later he complained of pain in the thigh and there was swelling, local heat, tenderness and fever. I incised the swelling and evacuated infected bloodclot.

It is hardly likely that a blood infection in the sense of a bacteriemia took place in this case. I would prefer the explanation of Bond (1921), who has drawn attention to the "return immigration" of leukocytes. Phagocytes ingest large numbers of organisms from the infected focus, transport them unkilled, and when the general resistance is lowered, allow them to become liberated and start a fresh focus of infection or a "recrudescence of the local sepsis." Early in the World War, Bond (1914) drew attention to the tendency of previously infected but healed war wounds to light up under the stimulus of a slight injury or a surgical operation. We can imagine phagocytes, loaded with bacteria, remaining quiescent in the scar tissue and set free by such influences as interfere with the local or even the general resistance. About six months ago I operated upon a woman for gall-bladder disease. The wound became infected, sloughed and a hernia developed. Recently, I reoperated on her for the hernia, and although extreme precautions were taken the wound is again infected, although only slightly. A second wound made to suspend the uterus has remained perfectly clean.

In some cases the normal skin may act as a focus of infection. Probably the normal inhabitant, the *Staphylococcus albus*, does not have much of a pathogenic action, but Schacter (1918) found hemolytic streptococci in 9 per cent of 89 persons with dirty skins and none at all on the bodies of 38 clean persons. It is well known that a hernia wound, the lower end of which often extends into the pubic hair region, will show a greater percentage of wound infection than an appendix wound.

Active infection in the skin may give rise to more serious infection than simple wound damage. Pimples may give rise to boils or carbuncles. The skin lesion may heal, but later an acute osteomyelitis may develop. Phemister (1922) has drawn attention to this type of focal infection and one of his cases is as follows:

A boy, aged fourteen years, had a boil on the right leg which was almost healed at the end of about ten days, when he developed extensive osteo-

myelitis of the entire shaft of the left tibia and upper half of the shaft of the right tibia. Arthritis followed in both knees and other joints and amputation of the left leg was necessary later. The organism found was the *Staphylococcus aureus*.

It will be seen, therefore, that focal infection interests the surgeon. We constantly urge the removal of the diseased appendix or gall-bladder, but I do not think we ourselves pay sufficient attention to preoperative removal of tonsillar or teeth infection. Everything points to the great influence of these organs. The removal of the infected tonsils should be preceded, especially in adults, by some local treatment directed against the crypts and space between the pillars and tonsil. The work of Fetterolf and Fox shows clearly that infected thrombi may act as septic emboli and produce lung abscess. There is also some risk attending the extraction of infected teeth, Blumer (1926) having reported several cases of general sepsis and the occurrence of infectious degenerative processes in patients suffering from chronic disease.

One final word: As Eusterman (1924) points out: "If the therapeutic results are disappointing, either the removal of demonstrable foci has been incomplete, coexisting infections have been left intact or extreme anatomic changes have been brought about as the result of long-standing systemic infection."

FOCAL INFECTION FROM THE VIEWPOINT OF THE DENTAL SURGEON*

By WILLIAM A. JAQUETTE, D.D.S.

Osler has been credited with advising his student to "treat the patient as well as the disease." In a consideration of "treatment," the patient's welfare must be the *first* consideration, but we all know his wish cannot always be lightly ignored.

The physician is not in a position to know what terror the thought of the loss of teeth brings. To many people it is a confession of the fading of youth. All men dread that state, as pictured in *Ecclesiastes*.[1]

> In the days when the keepers of the house shall tremble, and strong men shall bow themselves, and the grinders cease because they are few, and those that look out of the windows be darkened.

The training of the early dentists was to conserve the comfort, appearance and function of the teeth. In later years, however, it has been proven that *some* conservation has too often been at the expense of the health of the owner of infected teeth.

There probably has been no successful attempt to evaluate the relative frequency or virulency of dental focal infection as compared with infection from other foci, but, in general, it is well to consider the *infection* or the infecting organism, and not the focus, then there can be no doubt as to the importance of dental focal infection.

The two dental fields most frequently involved are the periapical and the periodontal. Microörganisms may reach the periapical tissue, there to establish what becomes a focus of infection by the way of an infected tooth pulp, resulting from dental caries or tooth fracture, as well as by blood or lymph circulation from adjacent or distant parts. Infection by this latter channel, however, seldom if ever occurs if the tooth pulp be normal. When such infection does

* Read May 5, 1926.

occur, it is because the tooth is devitalized and presents an area of lowered resistance.

To more fully appreciate the two types of oral infection above mentioned, a short description of the relationship of the parts of each may be offered. Attention has been drawn more frequently to the so-called periapical infection than to the periodontal.

A very large percentage of periapical infection may be directly traced to caries with resultant pulp exposure. The pulp consists of bloodvessels, nerve and connective tissue and odontoblasts. After infection it may be the seat of a number of minute abscesses throughout, which eventually coalesce. The root end may have single or multiple foramina, and through these the organisms may reach the tissues beyond. It may be worth mentioning that the greatest portion of the tooth is dentin, with enamel covering that portion called the crown, the part exposed beyond the gum. The dentin of the root is covered with cementum, with its pericementum. This latter membrane, the alveolodental periosteum, is a fibrous tissue comparable to periosteum, having its fibers inserted into the cementum of the root on one side and into the alveolar bone on the other.

Appleton has summarized the bacteriological study of the periapical tissues of pulpless teeth.[2]

> Of the microörganisms that are either aërobic or possess the ability of living under either aërobic or anaërobic condition, the common pyogenic cocci are encountered with the greatest frequency.
>
> Of these cocci, the streptococci by far outnumber all other types, and the vast majority of the streptococci are members of the short-chained, alpha or so-called viridans group, characterized pathologically by a relatively low virulency.
>
> The alpha streptococci, isolated from periapical or, for that matter, from any oral infection, do not constitute a single well-defined entity of the basis of fermentation reactions or serological tests. Judged by these criteria, the streptococci of oral infection fall into many separate groups, and are not distinguishable from the alpha streptococci from the saliva, from the tonsils or from other localities, diseased or healthy, of the human body.

In considering infection of the periodontal type, we have here a severing of the fibers of the peridental membrane as a sequence to irritation, which may be mechanical or toxic or due to atrophy. The result of such destruction of the fibers is to exaggerate the gingival crevice or trough between the tooth root and free margin

of gum. This, then, becomes a perfect habitat for the proliferation of the organisms of the mouth.

In describing the inflammatory phenomena in oral tissues, Appleton states.[3]

> There is nothing peculiar in the inflammations which affect the gingivæ. The epithelialization of pyorrhetic pockets may be noted in passing as evidence of the proliferative phase. The "purpose" of this new epithelial layer is to protect the underlying connective tissues, but inasmuch as it prevents close approximation of fusion of the gingivæ to the surface of the denuded root, it interferes with the realization of a legitimate goal of dental treatment. The same condition is observable in fistulous tracts, which likewise required deëpithelialization in treatment.

With the above picture in mind, we may not consider lightly the probability of such infected crevices being dental foci. The bacteriological study of pyorrhea reveals no specific infecting organisms — those isolated being merely those present in the mouth, with quite a large latitude of variation.

A practical application of a study of dental focal infection requires us to consider what is to be done with teeth involved as either primary or secondary foci. Should all pulpless teeth be extracted? To the hundred percenters, Yes. Are, however, these extremists serving their patients best in all instances?

Because many pulpless teeth eventually become infected, have we the right to assume all do? Because they may be considered potentially a seat of infection, should we condemn many useful organs?

It may be of interest to know the practice of leading dentists throughout the country. At a session of the New York Academy of Dentistry, January, 1926, Dr. William Dwight Tracy reported on a questionnaire presenting opinions from teachers of clinical dentistry and practitioners. The report showed a wide range of opinion, but a large proportion agreed that for patients in good health where there was no clinical or *x*-ray evidence of infection, pulpless teeth should be retained and kept under special observation and be examined and *x*-rayed at definite intervals.[4]

For that type of patient who is sick — those suffering from other than an acute temporary ailment, in whom the physician has found marked evidence of sepsis or organic degeneration, and an honest effort is being made to eliminate *all* possible foci by checking the alimentary tract, the upper respiratory tract, tonsils, etc. — the

dentist should be willing to coöperate and in extreme cases surrender teeth against which it is not possible to find any evidence except they be pulpless, because it is a well-known fact that an *x*-ray negative tooth *may* prove to be infected. The number of pulpless teeth in one mouth must surely be significant, for it is accepted that each such tooth presents an area of lowered resistance. Further, anyone of them may become involved from its infected neighbor or from the upper respiratory tract. Sometimes it is impossible to secure a sterile root-canal dressing until other oral infection has been removed.

On what, then, do we build our hope in conserving *any* pulpless tooth?

In many instances we object to the term "dead tooth," the reason for which is that a pulpless tooth is not of necessity a dead tooth. If the only source of nourishment of a tooth was *via* the pulp, then a pulpless tooth would be a dead tooth. The pulp, with its blood supply, nourishes the dentin, but the cementum is supplied by the bloodvessels of the pericemental membrane.

As evidence, we have hypercementosis of the successive lamella of cementum. Grove and Davis have shown that in many teeth the *vital apical tissue* has *caused* calcification and blocking of all foramina The real difficulty besetting the dentist in caring for the pulpless tooth is not so much the sterilization of the root canal as its obliteration with some type of filling which completely seals it against further ingress of organisms and is nonirritating. It is common practice to secure negative dressings moistened by seepage from periapical fluids from canals that have been infected.

When it becomes possible to so block the apical foramen or multiple foramina through the root canal, the lumen of which is exceedingly small, and have the peridental membrane lay down further laminæ of cementum, that tooth will likely be innocuous.

In making a dental diagnosis on which to make recommendations, *all* of the evidence possible to accumulate, is needed. In dividing the information gained by "clinical evidence," and distinguishing it against that understood as *x*-ray evidence, the former is of by far the greater importace. The *x*-ray is valuable in that it shows root-canal fillings, the relative condition of the peridental membrane, the lamina dura or compact bone lining the alveolus. It may show radiolucence, but it cannot show infection. Radiolucence is commonly caused by infection, but is sometimes due to mechanical or

chemical irritants. The *x*-ray cannot show deviation in the shades of color of a tooth, either marked or slight, though such deviation may be the only evidence noted to suggest the tooth be pathological.

The *x*-ray may not show an abscessed tooth with fistulous drainage. This is of common experience. Again, the *x*-ray film may show an artificial crown on a vital tooth and arouse suspicion unjustly, and fail completely to identify the tooth with septic pulp under a perfect filling, or the one with pulp destruction due to trauma with no external break.

Should we extract all pyorrhetic teeth? Those situated in a well of pus, with a large proportion of both fibrous and bony support destroyed; such teeth should be at once removed. The presence of pus at the gingival margin *alone* does not make extraction necessary. By the curettage of the necrotic peridental fibers and epithelial lining of the pocket the removal of all irritants, mechanical and bacterial, and the relief of undue stresses, many teeth may be restored to health and service. Pyorrhetic teeth having vital pulps do seem to present a much more favorable field than do pulpless teeth with but slight periapical involvement.

When, however, the tooth is pulpless and *x*-ray negative but showing advanced periodontal infection, it must be considered unfavorable by reason of the contiguous infection which likely will involve periapical infection.

In endeavoring to speak for the dental profession, I feel that I may *assert* that it is the desire of most dentists to coöperate with the medical profession to conserve the health of the patient. We feel we need the same spirit of understanding of the dental problem.

The loss of teeth is an evil: It may be the lesser evil, but the restoration of function and facial symmetry is always difficult, often impossible. The extraction of one tooth removes one-thirty-second of the number, but by its removal the loss of one-fifth or more efficiency may result. The teeth that remain drift; they incline so the occlusal relationship with the inclined planes is lost. The mouth is then made more susceptible to further caries and also to periodontal disease. The very restorations for the lost teeth often increase the susceptibility to further oral disease.

Before we remove teeth let us make every effort to assure the necessity. We are sometimes asked to remove all possibly suspected teeth. Then, later, we learn the tonsils or some other foci are manifestly bad.

It may be of interest to read a part of a letter received by Dr. Harold Craigin, of Thomas W. Evans Institute, from Dr. Cook, formerly instructor at the institute, now working with the Mayo Clinic:

> At present we are working on a pulpless tooth filling or root-canal filling that will prove sterile, but have no conclusions to draw as yet. This work we are doing at a state institution and now have several hundred teeth with root canals under observation. Over one hundred root resections have been completed and are under observation.
>
> I have been impressed with one thing here—that the dental department is not radical. We have a reason for removing each tooth and can *prove* its injury to the patient's general health.

We believe no rule may be applied to all cases; that when the general health of the patient warrants it, in cases of doubt, we are justified in keeping some teeth under observation and at a later date make a new study.

When, however, the physician confers with the dentist and assures him that the condition of the patient's health is grave, that a comprehensive study is being made for all foci of infection, then the dentist must be willing to be more radical.

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2. Appleton: Bacterial Infection, Lea & Febiger, 1925, p. 388.
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DISCUSSION ON THE PAPERS OF DRS. KOLMER, PEPPER, DE SCHWEINITZ, MÜLLER AND JACQUETTE

Dr. J. M. Anders: In the first place we have had a clear view of a many-sided condition and one that should assist us all. My remarks, however, will be limited to the medical aspect of the subject. Dr. Pepper made reference to the fact that two large groups of cases, practically considered, occur: the one in which distinct metastatic abscesses or lesions occur and the other in which this does not take place. In other words, there is a large group which we medical men meet with in which there is no evidence of metastatic development anywhere to be found in the body and these—at least many of them—present very ill-defined and vague symptoms. In some of them I have observed that the principal symptoms are nervous

in character, there is more or less nervous headache and palpitation and dyspnea on slight exertion. A routine blood examination shows in many of them a decided grade of symptomatic anemia. Now these are the cases, doubtless, in which the bacteria do not set up secondary foci in the body, but are overcome in the blood stream by what has been referred to tonight by Dr. Kolmer's paper as "the bactericidal property of the blood." The indefinite group of symptoms, some of which are due to the associated anemia to which I have referred, do not as a rule give rise to suspicion of focal infection, but I would like to urge that when such symptoms as are described do not yield to the usual measures of treatment within a reasonable length of time, focal infection should be suspected and search made for foci of infection. I have discovered them in these instances on examination of the teeth, the tonsils and the sinuses, as well as elsewhere. It is principally for the reason that a cure is sometimes, at least, rapidly effected in this class of cases that I want to call special attention to it. For example, if the primary focus be removed, the symptoms are very easily controlled and may rapidly disappear; and one can readily see the reason for this in the absence of secondary developments elsewhere in the body. I am sure that the group is larger than might at first sight appear, if I may base this remark upon personal experience and observation.

Dr. Jay F. Schamberg: The various essayists of the evening are to be congratulated upon their excellent presentation of a very difficult subject, a subject concerning which no dogmatic assertions can be made. We are all familiar with the fact that there are perfectly healthy individuals who may present, as has been stated, focal infections without apparent harm. The question is really one of degree of resisting power upon the part of the patient. We harbor upon mucous membranes and upon the skin certain pathogenic organisms; I am inclined to believe from certain investigations made in our Institute that we harbor streptococci upon the skin in a larger percentage of cases than is stated by the investigator quoted by Dr. Müller. Some years ago the late Dr. Gildersleeve and I called attention to comparative cultural findings in the throat in scarlet fever and in normal subjects. We found 92 per cent of scarlet-fever patients whose throats were positive, but we also found 82 per cent of University students who had streptococci on their tonsils, and these killed experimental animals with the same facility that the scarlet-fever streptococci did. The question at issue is whether a given focal infection is responsible for a presenting morbid condition. The coincidence of focal infection and a morbid condition in the same individual does not of necessity indicate cause and effect.

As regards cutaneous diseases, perhaps the most definite conditions due to focal infection are furunculosis and carbunculosis; we undoubtedly see patients with recurring furuncles or carbuncles which are due to infection about the tonsils, teeth, prostate, etc., and the removal of these infections will commonly lead to the disappearance and cessation of the cutaneous complication; we may, however, often improve or cure the condition without removal of the original focus of infection by local treatment or by increasing the patient's resisting power by vaccines or by nonspecific

protein injections. The relief may be permanent or may be temporary; it commonly is temporary until the ultimate cause of the condition is removed. It is interesting, however, that one can raise the resisting power of the individual to such an extent that the cutaneous infections are prevented. This is an interesting commentary not only on the existence of infection, but on the manner in which it is tolerated. We know that a man may harbor pneumococci in his throat and be immune until exposed to cold and wet, and then his resisting power is lowered and he takes pneumonia. The balance of power is changed and instead of his being the master, the organisms become dominant. May this not take place in focal infections?

Dr. Pepper has well expressed the proper medical attitude toward this subject in maintaining a considerable degree of mental reservation as to the limitations of the effect of focal infections. We have all seen cases in which the removal of the discovered focal infection removed the clinical condition under study, but I think in a much higher percentage of cases we have observed that the removal of the focal infection failed to accomplish this purpose.

Dr. Astley P. C. Ashhurst: In this symposium on focal infections, I have heard little discussion about focal reaction. The diagnosis of tuberculosis of the joints, especially in adults, sometimes is very difficult to make, and surgeons are inclined to use the hypodermic tuberculin test (O.T.). Patients may have malaise and fever between the eighteenth and twenty-second hour following the injection, and this is considered evidence that the joint affected is tuberculous; but especially characteristic is a focal reaction in the suspected joint in addition to the general reaction. Now, if you have a patient with a localized lesion (*e. g.*, arthritis) which you suspect of being due to some other focus (for instance, a diseased tooth), and if removal of that focus causes a focal reaction, can you assume with any more confidence than without such reaction that the focal infection is the cause of the local lesion?

OBSERVATIONS ON THE HEART IN THYROID DISEASE*

By C. C. WOLFERTH, M.D.

AND

EDWARD ROSE, M.D. (By Invitation)

The frequency of cardiac disorders in hyperthyroidism was recognized by the earliest observers of the disease. Whether it be in hyperplastic toxic (exophthalmic) goiter or in the so-called toxic adenoma of the thyroid, tachycardia and increased forcefulness of heart action are almost constant; dyspnea, palpitation and throbbing of the great vessels of the neck are of frequent occurrence. The great majority of patients with hyperthyroidism do not present further evidence of cardiac derangement, but in a smaller group cardiac symptoms may dominate the clinical picture. In the latter, cardiac incompetence constitutes a therapeutic problem of major importance and the condition of the heart may be the deciding factor in swinging the balance toward recovery or death.

There is a striking lack of agreement as to the frequency and importance of actual pathological changes in the so-called goiter heart. Fatty infiltration, small scars and focal necroses and interstitial or perivascular cellular infiltration are found at times. Whether such changes are more frequent in the hearts of patients dead of thyrotoxicosis than in those dying of other causes at similar ages cannot be stated. Certainly specific lesions in the heart of hyperthyroidism have not been found and often no significant changes whatever are discovered. Enlargement of the cardiac outline is often found during life, but how often this may be due to dilatation of the chambers without myocardial hypertrophy is uncertain. It has been stated that the proportion between myocardial weight and body weight is seldom altered in hyperthyroidism.

The manner in which the cardiac changes are brought about

* Read June 2, 1926.

remains obscure. It has been variously suggested that the toxic product of the thyroid may act directly on the myocardium; that this noxious agent may increase the susceptibility of the heart muscle to coexistent infectious processes or lessen its ability to withstand the added strain thrown upon it by the increased demands of the body for oxygen, and, finally, that the entire process is simply the result of work hypertrophy consequent to the increased metabolic rate. Unquestionably the increased oxygen consumption and carbon dioxide production characteristic of hyperthyroidism call for an increased bloodflow even though the body be at rest, and this must be met by increased cardiac activity. Whether these factors alone are concerned or whether there is, in addition, a specific action of abnormal thyroid secretion must remain unanswered for the present. Some writers have postulated the theory which assumes the cardiac changes to be the result of excessive sympathetic stimulation, but this too must remain undecided.

In the earlier stages of hyperthyroidism the size of the heart is seldom increased. Tachycardia is an early and constant finding, and is often, although not always, proportional to the degree of elevation of the basal metabolic rate. Slight dyspnea, palpitation and precordial discomfort, with consciousness of the heart action and throbbing of the great vessels of the neck are frequent. Systolic murmurs of varying intensity and quality may be heard, usually over the apex, later at the base as well, or over the entire cardiac area. The sounds are increased in intensity, especially the first sound, which may be prolonged. As Kerr and Hensel have well put it, however, the heart sounds give an impression of hurried ineffectiveness. The apical impulse become forcible and diffuse, and, as dilatation or hypertrophy occurs, shifts downward and to the left. There is a strong pulsation of the neck vessels. The pulse-pressure is increased, due usually to an elevation of the systolic and depression of the diastolic pressure, so that the radial pulse may resemble that seen in aortic regurgitation and capillary pulsation may be demonstrable. Expansile pulsation of the thyroid gland from changes in its blood volume with each systole may be seen, or it may transmit the pulsation to the carotid vessels. The rate of bloodflow through the thyroid may be increased five- or tenfold; the volume of cardiac output per beat is little if any increased.

Arrhythmias are frequent and may be of any type: Sinus

arrhythmia, extrasystoles, paroxysmal tachycardia, auricular flutter or partial heart-block occasionally occur, but auricular fibrillation is by far most important. It may be continuous or paroxysmal; it seldom occurs early in the disease. Heart-block, if not due to digitalis, denotes serious myocardial affection, but paroxysmal flutter or fibrillation may occur for a long time without any other evidence of circulatory weakness. The reported frequency of this type of arrhythmia varies: Kerr and Hensel observed it in 30 per cent of their toxic cases; Hamilton mentions an incidence of 10 per cent and Willius about 8 per cent. The incidence would, of course, depend upon the stage in which the patients were observed, but transient periods of arrhythmia are probably more frequent than is generally supposed. If the thyrotoxicosis be relieved, spontaneously or by treatment, the arrhythmia may permanently disappear, especially if it has not been long existent. Paroxysmal fibrillation seems to be almost always abolished by successful surgical treatment; the established type may revert spontaneously to normal rhythm.

Even advanced cardiovascular insufficiency may be relieved by treatment of the hyperthyroid state, with restoration of normal rate, decrease of the area of cardiac dulness, relief of distressing symptoms and disappearance of peripheral evidences of heart failure. Such improvement depends chiefly upon the stage in which treatment is instituted and the type of treatment employed. One of the principal advantages of surgical treatment appears to be its greater efficiency in relieving cardiovascular embarrassment. By a successful operation, at one stroke, the patient is relieved of the cause of his general intoxication and the link in the chain which causes excessive cardiac stimulation is broken.

If the thyrotoxicosis persists, cardiovascular insufficiency is progressive. The previously mentioned symptoms increase in intensity, the cardiac outline becomes more and more increased, previously paroxysmal arrhythmias may become continuous, the patient becomes a cardiac cripple and the scene is terminated by the typical picture of congestive heart failure. On the other hand, there may be seen cases in which the evidences of cardiovascular failure are not striking, yet in which the occurrence of a so-called thyroid crisis or storm results in circulatory collapse and death. Whatever be the cause of the exacerbation of thyroid intoxication, it would seem that, in some of these patients at least, death is

attributable to cardiovascular weakness though congestive heart failure does not occur.

The writers wished to determine the frequency and clinical nature of cardiac disturbances in thyroid diseases at the University Hospital and the response of such disorders to treatment, particularly in connection with operation. We have, therefore, examined the records of 201 consecutive admissions of patients with thyroid disease to Dr. Frazier's surgical service. This group included both toxic and nontoxic goiters. (All of the patients to be referred to below have been under the personal observation of one or the other of us, and in practically all instances were followed throughout their stay in the hospital). We wish to take this opportunity to thank Dr. Frazier for his kindness and coöperation in permitting us to utilize this material.

In the 201 admissions there were 18 patients (all with toxic goiters) who presented sufficient evidence of heart disease to warrant special preoperative medical observation and treatment. All of these were subjected to operative procedure—15 survived and 3 died, a mortality of 16.6 per cent, which is in striking contrast to the average general mortality in thyroid operations of about 1 per cent. Only 2 of the 3 deaths, however, were attributable to heart failure. In the cases that recovered, ligation alone was performed 5 times, ligation followed by partial thyroidectomy twice, and partial thyroidectomy without previous ligation 8 times. One of the fatalities followed ligation and the two due to heart failure occurred twenty-four and thirty-six hours, respectively, after subtotal thyroidectomy.

Seventeen of the patients were females. The ages ranged from thirty-five to sixty-two years, with 11 in the fifth decade. The duration of symptoms varied from less than one year to seven years—the largest group (8 cases) had had symptoms less than one year, and both the cardiac fatalities occurred in this group. The basal metabolic rate was + 40 per cent or above in the great majority on admission, and was + 65 per cent or above in the 3 fatal cases. The figures on weight loss were not accurate enough to permit of analysis, but the losses in general were marked, although the degree of weight loss could not be correlated with the basal metabolic rate on admission.

Cardiac enlargement to percussion was noted as absent in 3 cases, slight in 1, moderate in 7 and marked in 7. Compensation was preserved in 8 cases; slight decompensation was noted in 5, moderate

in 3 and marked in 2. The heart rhythm was normal in 6 instances, intermittent flutter was present in 1 case, paroxysmal fibrillation in 4 and established fibrillation in 7. All cases showed a marked tachycardia, either constant or intermittent. The 3 fatal cases showed moderate enlargement to percussion; all showed paroxysmal fibrillation; 2 had compensation preserved, the other showed evidence of slight decompensation (pretibial edema). Of the cases with arrhythmia, 4 had definite decompensation and 4 only slight compensation. Only 2 patients with decompensation showed normal rhythm.

In the 2 cases in which death seemed due to cardiac failure, marked tachycardia followed by collapse occurred twenty-four and thirty-six hours, respectively, after subtotal thyroidectomy. Auricular fibrillation recurred in 1 case. Congestive heart failure was not observed in any of the cases after operation. In 2 of the patients who died we felt that death was due, at least in part, to the inability of the cardiovascular system to withstand the strain of postoperative exacerbation of thyroid intoxication. Persistence of arrhythmia was noted after operation in 5 instances, disappearance in 5, and in 2 the records are incomplete. The period of postoperative observation varied in this group of patients from a few weeks to two years.

Treatment consisted of high caloric diets, complete rest, sedatives as required, digitalis (usually in the powdered form), and, in the cases of true hyperplastic toxic goiter, compound solution of iodin. Digitalis was apparently of no value in reducing the rate in cases with normal rhythm. There was no evidence that it prevented the occurrence of paroxysmal fibrillation either pre- or postoperatively or that it aided in restoring normal rhythm in fibrillators. This observation is not in accord with those of others (Kerr and Hensel), who have concluded that fibrillation in thyroid disease may be prevented at times by smaller amounts of digitalis than is required by ordinary cases. We did find that digitalis had a tendency to control the ventricular rate during fibrillation if a sufficient amount were given, although the dosage required varied widely. Some thyrotoxic patients exhibit a high tolerance to this drug—1 of our patients received 4.5 gm. (67 gr.) of powdered digitalis in nineteen days without any evidence of toxic digitalis effect. Compound solution of iodin was of distinct value in the cases in which it was used (the exophthalmic type) in lowering the basal metabolic rate and thus in improving the general condition; it also seemed to exert

an indirectly beneficial effect on the circulation, probably by decreasing the work required of the heart. The multiplicity of factors involved in the treatment of these cases renders the evaluation of each difficult; rest, the use of digitalis, and in selected cases Lugol's solution, are all of importance where there is evidence of cardiac incompetence. Prolonged treatment along these lines will in many instances bring cases with apparently hopeless cardiac degeneration to the point where operation may be performed with reasonable safety and their chances for permanent relief increased. One of our cases in particular illustrates this point:

Mrs. M. S., aged forty-eight years, was admitted May 4, 1923, with a nodular thyroid enlargement which had shown signs of toxicity for about a year. For five months there had been gradually increasing dyspnea, peripheral edema and at times hemoptysis. Palpitation, cardiac discomfort and fatigue had been marked. Examination disclosed exophthalmos, nodular left-sided thyroid enlargement (adenomatous) and evidence of loss of weight. There was marked dyspnea, pulmonary congestion, signs of pleural and abdominal effusion and edema of the legs and back. There was a diffuse pulsation over the area of cardiac dulness, which was much increased in extent, the left border being found at the anterior axillary line; jugular pulsation was marked, the ventricular rate was very rapid and totally irregular, with a considerable pulse deficit, the heart sounds varying widely in intensity; a loud systolic murmur was audible over the entire precordium. The basal metabolic rate was + 59 per cent.

The patient was seen by Dr. Wolferth on the day after admission and powdered digitalis, 4 gr. (gm. 0.24) daily, ordered. In addition, her fluid intake was curtailed and she received a diuretic (theoscin) for a few days and sedatives as indicated. By May 12, a week later, the ventricular rate was still 140 and the auricular fibrillation persisted, with little if any decrease of the edema, although the dyspnea was perhaps less troublesome. The same dosage of digitalis was continued, and by May 20 her condition was much improved—the ventricular rate was down to 90, there was some irregularity, consisting mostly of extrasystoles, the edema was much less extensive and the dysphea was almost completely relieved. Her condition was considered satisfactory for operation, and on May 23 the adenoma was enucleated by Dr. Frazier. Her heart-rate and rhythm remained perfectly normal after the operation, despite a secondary hemorrhage, and on May 25 the digitalis was stopped because of a pulsus bigeminus. Her convalescence was uneventful and she left the hospital June 17 in excellent condition.

In summary it may be said:

Severe heart disorders have been observed in approximately 9 per cent of our patients with thyroid disease, but occurred exclusively in cases of toxic goiter.

Cardiac failure in hyperthyroidism is in the majority of instances associated with auricular fibrillation or flutter.

Cardiac complications adversely affect the prognosis in hyperthyroidism and markedly increase the operative mortality.

Circulatory insufficiency may often be relieved by careful and prolonged treatment and operation successfully performed with good expectation of causing the permanent disappearance of cardiac symptoms.

An important advantage of subtotal thyroidectomy appears to be its efficacy in checking the progress of cardiovascular derangements.

Certain cases which present little evidences of circulatory weakness may succumb to cardiovascular collapse under the added strain of operation or postoperative thyroid crisis.

The most important factors in the management of heart disease in hyperthyroidism are rest, the use of sedatives, digitalis which is of most value when fibrillation is present—and in selected cases compound solution of iodin.

Certain cases of thyrotoxicosis exhibit an increased tolerance to digitalis.

Iodin is not only of value in reducing the basal metabolic rate, but also appears to indirectly improve the circulation by decreasing the work required of the heart.

DISCUSSION

Dr. Charles H. Frazier: We regard it as a matter of very considerable importance in the management of the thyroid case to have the advice of our medical confrères. As a matter of fact, as the Thyroid Clinic at the University Hospital is organized, practically all cases are seen by the medical consultants of the Thyroid Clinic staff. Contrary to what is often stated, not very much importance is attached to tachycardia in gauging toxicity and selecting the type of operation. We have come now to disregard, to a very considerable degree at least, the pulse-rate alone as a measure of toxicity. So far as cardiac manifestations of toxic goiter are concerned, our anxiety is aroused chiefly when there are signs of myocardial disease, of cardiac decompensation and more particularly of arrhythmia. So, too, I think, contrary to what is usually stated and thought, the patients who present the greater hazard are not the true exophthalmic goiters but the toxic adenomata, and there is a reason for this. The toxic adenoma patient is usually more advanced in years, often over forty, and because of the

patient's age and the duration of the lesion one more often finds signs of myocardial degeneration. The lesson to be learned, from what Dr. Rose and Dr. Wolferth have presented tonight, is obvious. There is a definite risk in operating late in the course of the disease and none in the early stages. The 3 fatalities they selected for their discussion this evening represent the only fatalities in our last 300 cases. I was a little surprised that they stated there were only 18 evidences of cardiovascular disease in this series, but I think in his closing remarks Dr. Rose qualified this statement by saying these 18 cases represented the *severe* cases. I am sure there are many more with moderate signs of circulatory embarrassment, such as shortness of breath and edema of extremities. The time has come, I think, when we owe it to the patient to advise operation at a reasonably early stage, because we can state then, without fear of contradiction, that at that time there is no risk attending a thyroidectomy. The 3 fatal cases to which Dr. Rose and Dr. Wolferth referred had been suffering for from six to seven years. As presented to us they were recognized as grave hazards, and even though several weeks were given over to preparatory treatment in these 3 fatal cases disaster could not be averted. I wish in closing, therefore, to make an appeal for early resort to operation in all cases of toxic goiter. Just as the general surgeon may decline to operate in a case of established peritonitis secondary to appendicitis, so in the thyroid clinic the surgeon may be justified in declining to operate in the face of what seems to be certain disaster.

Dr. Hobart A. Hare: The Chair is well aware it is not within his province to speak, nevertheless he would ask this question: I think it is generally considered the high metabolic rate in hyperthyroidism necessarily involves an increased amount of heat production in the body. The best therapeutics is to give water very freely indeed. I understood the essayist to limit the amount of water intake in twenty-four hours. The other point Dr. Frazier has already alluded to, that no one can have hyperthyroidism over a long period of time without other organs of the body suffering as a result of the hyperthyroidism, just as a child cannot have diphtheria without other organs suffering. Of course, the difference in rapidity of change is very great. I did not notice—perhaps I did not pay attention—that the essayists gave us any direct information as to the vascular system or kidneys in their paper. That would have had some interest as bearing on the question of danger of operating on these cases, and might have some interest in determining how soon, after hyperthyroidism develops, degeneration of other organs occurs. The chief point I wish to emphasize is to be either set right, or set wrong, on the question whether I am correct as to water intake in this condition. Of course where there is a cardiac dropsy, as mentioned in one case, then it may be wise to limit water intake, but in general terms I give water in all cases.

Dr. Wolferth (closing): Dr. Rose and I do not favor diminution of water intake in hyperthyroidism, but quite the contrary. These patients have an augmented water metabolism, and I certainly agree with Dr. Hare

that they need more water than the normal individual. The one exception we make is the limitation of water intake during attempts to dehydrate dropsical patients.

Routine examinations of urine were made in all patients, and, in practically all, phenolsulphonephthalein tests were made. If these showed anything abnormal, blood-urea studies were also made. While some of our cases showed renal disturbances, these were not a conspicuous feature.

A point Dr. Rose and I wish to emphasize particularly is the fact that in the thyrotoxic fibrillating heart the dosage of digitalis is of great importance. One will find statements by some authors that digitalis is of little or no value in controlling the ventricular rate in auricular fibrillation of thyroid origin. We were able to prove conclusively that ventricular rate can be controlled if sufficient digitalis be given. We have conjectured that with the increased metabolic rate the metabolism of digitalis—at least the rate of excretion—might likewise be increased and for this reason larger doses required.

The other point in connection with digitalis we wish to make is that no value in controlling tachycardia was noted following its use in patients with normal rhythm. We are not, however, prepared to say that digitalis has no value in patients with normal rhythm.

Dr. Frazier stated that in some patients with severe thyroid disease, operation must be refused. The patient with congestive heart failure, cited by Dr. Rose, was one of these. The condition of this patient on admission to the hospital precluded operation as entirely too hazardous. Nevertheless, after prolonged treatment according to the plan outlined by Dr. Rose, so much improvement resulted that operation became feasible and an excellent result obtained. This patient is cited as a sample of a small group headed for almost certain death if treatment is exclusively medical or surgical, but most of whom may be saved by close coöperation between internist and surgeon.

Dr. Rose (closing): I am very sorry to have given the impression that we curtail fluid intake in all of the cases referred to. It was only in cases with severe cardiac failure, as evidenced by the presence of serous collections in the pleural spaces, peritoneum, and perhaps pericardium, that the intake of fluid was curtailed. In such instances we thought that the elimination of fluid was one of the primary therapeutic indications.

As Dr. Frazier remarked in his discussion, there were far more than 18 patients in this series who presented transient arrhythmia or slight evidence of cardiac insufficiency. These 18 cases are only those which presented sufficiently severe evidence of cardiac involvement to warrant special observation and treatment.

THE TREATMENT OF ACUTE EARLY SYPHILIS*

By SIGMUND S. GREENBAUM, B.S., M.D.

By acute early syphilis, we mean infection by the *Spirochæta pallida* from its incipiency up to and including the cutaneous manifestations of the generally known secondary stage. We will not here discuss early congenital syphilis, nor will we here include those manifestations upon the skin and mucous membrane which develop from six months to three years after the initial infection. These latter, known as "late secondaries," include recurrent roseola, recurrent follicular and other papular syphilides, neurosyphilides, erosive syphilides or mucous patches, and all may, at least from a clinical and prognostic aspect, be properly grouped under the term, chronic early syphilis.

With but little doubt, it may be stated at the outset, that the discovery of the causative organism of syphilis, but more particularly of the value of the dark-field microscope in its detection, as well as the introduction of the new complement-fixation and precipitation tests in the serum diagnosis of the disease, has enabled us to definitely place syphilis among the curable diseases. As with cancer so with syphilis, the earlier the diagnosis the more effective the treatment and the more certain the cure. Most syphilographers are now agreed that reinfection no longer constitutes the only criterion of this, and that superinfection, although experimentally possible, is only clinically proven in certain congenital syphilitics in whom the maternally transmitted *Spirochæta pallida* has become less virulent.

Therapeutically considered, acute early syphilis consists of three periods: An incubation period, during which there are neither cutaneous nor serologic manifestations; a second period, when the

* Read October 6, 1926.

chancre develops, but as yet without discoverable serologic changes; and a third period, of the chancre with discoverable serologic changes by means of the Kolmer or Kahn tests, and with or without the generalized exanthem of the clinically described secondary stage. With the development of the chancre, it makes no difference therapeutically whether the secondary eruption is present or not, once the serum has become positive to complement-fixation or precipitation tests, or both, the treatment is the same. Syphilis properly treated during any of these periods will not likely become a nervous, visceral, bone or pediatric problem.

Abortive Treatment. Abortive treatment, in the sense here used, must be differentiated from prophylactic treatment excepting as this term is used to indicate treatment immediately or shortly after infection. For practical purposes the abortive treatment should be designated as immediate, *i. e.*, within twenty-four hours, and late, *i. e.*, after that time. Irrespective of the mode of contact, infection by the *Spirochæta pallida* can only be presumed to have occurred. Some of these contacts are more certain than others, as for example, the physician who pricks himself with an implement just used on an active syphilitic lesion. However, by far the greater number belong to the group of merely suspicious contacts. Immediate abortive treatment is certainly indicated in the first instance. In the others, if abortive treatment is desired, it should be given with but a single exception, not later than twenty-four hours after exposure. If more than twenty-four hours has elapsed, it is much wiser, unless a full series each of arsphenamine or neoarsphenamine and of bismuth are given, to await developments. Otherwise one runs the risk of indefinitely concealing the presence of the infection, as insufficient treatment has been shown to produce, both experimentally and clinically. By awaiting developments is meant, not only for the appearance of a chancre—this may never be discovered, in the case of a woman, for example—but serologic studies at three-day intervals for six weeks after the incubation period of the disease has passed. In this connection it is well to recall a fact which many forget, namely, that discoverable blood changes never occur until the chancre appears, so that serologic studies are utterly useless immediately or shortly after exposure. The single exception, spoken of above, refers to the person, not infrequently a wife, innocently and repeatedly exposed to infection for one to two weeks, by another with an undiagnosed chancre.

In such instances, where contamination is almost certain, late abortive treatment in the form of a full series of neoarsphenamine and a full series of bismuth is indicated. Under no circumstances is such a patient to be simply given one or several injections of an arsenical and thus lull both patient and physician into a false sense of security.

Having decided that a case is admissible for immediate abortive treatment, the following is advised: Local use of 33 per cent calomel ointment wherever possible, if exposure is less than three hours. In addition, arsphenamine 1.8 gm. or neoarsphenamine 2.7 gm. in divided doses, as closely spaced as the patient's tolerance will permit, +60 cg. of bismuth tartrate in divided 200-mg. doses, intramuscularly, will surely abort the disease.

Following both immediate and late abortive treatment, observation, consisting of serologic studies every three months over a period of one year, is advisable. In these cases a spinal-fluid examination may be dispensed with, particularly if the blood tests are complement-fixation and precipitation ones.

Sufficient instances of reinfection have been described as to make it certain that abortive treatment cures syphilis provided the dosage given takes into account the duration of the incubating organism.

Seronegative Chancre. *Curative Treatment.* There are a few, supported by some experimental and perhaps clinical evidence, who feel that no treatment should be given until the invading organism has had time to stimulate all the antisyphilitic bodies of which it is capable. These "watchful waiters" even encourage dermal manifestations. At the present time, however, the concensus of opinion is that once a chancre has developed and been diagnosed as such, immediate treatment is indicated. Since the greatest chances of cure are obtainable during the seronegative period, it becomes urgent that all genital erosions and ulcerations be examined ultramicroscopically. The earlier the diagnosis, the more certain the cure.

Seronegative chancres are either treated intensively or, what may be termed, subintensively, which constitutes the method of procedure of most syphilographers. Irrespective of the method used, one of the arsenobenzenes, undoubtedly the best of the antisyphilitic remedies at our command, constitutes the first agent used.

The intensive treatment consisting of daily intravenous injec-

tions of large doses of arsphenamine or neoarsphenamine, is not to be advised, because cure can be obtained with greater safety by the subintensive method; because large doses at twenty-four-hour intervals is too short a time to estimate the drug tolerance of a patient; because we possess no antidotal drugs once the body has become supersaturated with arsenicals; because death from as low a dose as 150 mg. of neoarsphenamine has been described; because the production of acute arsenicism or, as it may be considered, hyperparasitotropic or organotropic effect does not always lead to complete annihilation of all spirochetes, as visceral, cutaneous and neurosyphilitic lesions have been noted subsequent to such accidents. Furthermore, it is believed by a few that the increase in incidence of neurosyphilis is the result of such early intensive treatment.

The subintensive method constitutes the method of choice, but it must be constantly kept in mind that the total dosage, although important, is less so than the size of the dosage at each injection. Reliance, either in this or the next stage, must not be exclusively placed upon the arsenicals, despite the fact that it has been shown that in many instances they alone suffice. In short, large doses of arsphenamine or neoarsphenamine rapidly reached by increasing the earlier testing doses, plus bismuth or mercurial injections, without any rest periods, constitute the subintensive method.

The diagnosis made and the patient found to be in good physical condition, 150 mg. of arsphenamine or neoarsphenamine are immediately given intravenously. In tolerant patients, the dose at two-, then four-, five- and seven-day intervals should be rapidly increased to a full dose of 60 cg. of arsphenamine or 90 cg. of neoarsphenamine to a total of 4.8 gm. of arsphenamine or 10.8 gm. of neoarsphenamine. This total dosage is larger than that usually recommended abroad, but is based upon unpublished studies of the total dosage, when maximum doses are given at each injection, required to reduce the strongly positive complement-fixation reaction of the secondary stage to negativity.

Immediately following the arsenical series, intramuscular injections of at least a 60 per cent bismuth is divided into 100- to 200-mg. doses to the total of 2 to 3 gm. are given.

The above comprises an arsenical and bismuth series, and both should be repeated. This treatment lasts about one year. Serologic studies are made after the first arsenical series and is almost

certain to be negative; again at the end of the second arsenical and bismuth series; then every three months for a year, with, finally, a spinal-fluid examination and provocative test. The blood will have been negative for over one and a half years.

Seropositive Chancre with or without Secondary Manifestations. Treatment for this stage differs from treatment of the seronegative stage only in duration. Instead of two series of the arsenical and the bismuth, three series are advised, with the last bismuth course rejected for a course of mercury in the form of innuctions or injections. Cure is based upon a complement-fixation and a precipitation reaction which has been persistently negative for two years and includes provocatory stimulation and a spinal fluid examination.

Although I have given a rather standardized outline of the treatment of acute syphilis, all syphilographers recognize the fact that there are a number of variable conditions and factors governing the administration of these drugs. Such conditions as drug intolerance and drug resistance are particularly important and it is admitted that the treatment will have to be varied to suit these individual conditions.

However, this standardized outline serves a twofold purpose: (1) It gives the general practioner a practical working plan and (2) I hope that it will, in part, at least clarify the chaotic situation which has arisen as a result of the introduction of numerous new preparations of arsenic and bismuth, whose indications and therapeutic values have, besides, been insufficiently studied.

THE DIAGNOSIS AND TREATMENT OF CARDIO-VASCULAR AND VISCERAL SYPHILIS*

By JOHN H. STOKES, M.D.

I OPEN with an apparent truism that the successful diagnosis and treatment of all special aspects of late syphilis lies in the detection and effective treatment of early syphilis in general. In no field has this principle greater need of appreciation than in that of cardiovascular syphilis. Even our earliest clinical recognition of vascular involvement in this disease is unhappily comparatively late. Generations of bowing at the shrine of the spectacular in physical diagnosis has made it still later. The cor bovinum and the hatbox aneurysm, the thrill, the buzzing and whirring, the heave, the sound of the pistol-shot, have blunted the sense of the practitioner for the still small signs and symptoms of preventable onsets of syphilitic cardiovascular disease. Until teacher and textbook turn from the parading of consequences before the student of medicine, to urge him to devote his energies to anticipatory watching, to habitual suspiciousness and observation, and to an acute analysis and ferretting out of veiled beginnings, syphilis of the heart and aorta will remain the great burying-ground of the disease and the clinician's perpetual Waterloo at the hands of the post-mortem pathologist.

Diagnostic cardiovascular syphilology needs, paradoxically, as I have seen it, both a renewed allegiance to and liberation from the bondage of the blood Wassermann test. The routine application of the test in every medical examination would immeasurably further the cause of prevention in syphilitic heart disease by the detection of syphilis in spurious latency. But in vascular syphilis there is reason to believe that the positive efficiency of the average blood Wassermann test ranges only between 60 and 80 per cent. As much as a third to a fifth of cardiovascular syphilis may there-

* Read October 6, 1926.

fore escape the Wassermann-bound clinician. The earlier the case, one would think, the more effective the blood test. Yet I have seen repeated examples of serological negativity within the first five to ten years, with indubitable lesions, and have watched some of them develop in patients under my own observation and treatment from the time of their secondary eruptions.

If the physician is to be deprived of his serological touchstone, by what signs shall he interpret? Paradoxically again, by the sign of symptoms, for the onset of syphilitic cardiovascular disease is earliest suggested by symptoms rather than signs. In the patient who has had syphilis, paroxysmal dyspnea, especially nocturnal; the sense of precordial stress, or later, pain; palpitation and dyspepsia form a tetrad which, though not distinctive of the syphilitic heart, must at once direct attention to the heart in the syphilitic. Pain, dyspnea, hoarseness, sometimes transitory, and cough, rank first in the symptomatology of aneurysm. The carefully taken history is therefore invaluable in interpretation of the cardiovascular situation in a patient with syphilis. To the physical signs, however, the physician must turn for diagnostic confirmation and amplification. One who watches the progress of cardiac syphilis through clinical practice, interpreting aftermaths, soon comes perhaps to overstress the earliest signs the ringing aortic second sound and, later, the early systolic murmur at the aortic area transmitted along sternum and often heard best for a time at the xiphoid process. Slight hypertension, slight broadening of the base of the heart, slight accentuation and outward displacement of the apex impulse are the neglected or minimized warnings of many syphilitic cardiovascular disasters. In the patient whose syphilitic infection is a matter of question, no history of the disease is obtained in 33 per cent in our experience. The provocative procedure may be dangerous, even fatal. The repetition of the blood test for a number of times adds 10 to 15 per cent to the possibility of a positive: 54 per cent of my patients showed evidence of concomitant neurosyphilis and 40 per cent had abnormal spinal fluids—an important clue therefore in identifying the presence of a concealed syphilis but unfortunately difficult to popularize because the early cardiovascular case presents so little to make an intensive investigation seem necessary to either patient or physician. Too ready an invocation of "rheumatism" as an explanation of Wassermann-negative aortic lesions seems to me a criticizable feature of present-day practice.

Syphilis itself, for one thing, has too much "rheumatic" symptomatology to make the history wholly trustworthy. The syphilis-conscious physician, confronted with the symptoms and signs of early aortitis, in the absence of indubitable evidence but the presence of suspicion, must therefore, I believe, proceed to *therapeutic test* much oftener than has been the fashion heretofore, for the issue at stake is literally the patient's life, which will not be in the least endangered under ordinary conditions by careful treatment for syphilis.

The *x*-ray and the electrocardiograph hold positions especially deserving of critical consideration. The early roentgenological signs of aortic syphilis have seemed disappointing to many English and American observers. Within the first decade I have rarely seen the fluoroscopic picture give the clue to a diagnosis of aortitis or alter the decision of an alert clinician after a thorough study of the patient's signs. When aneurysmal dilatation is well established the *x*-ray of course becomes more essential, but still hardly more so than skilled percussion and palpation, when it is practised.

In fully developed aneurysm, *x*-ray diagnosis falls afoul of the problem of the mediastinal mass. The nonpulsating aneurysm is mistaken repeatedly for tumor. Yet in my own observation a therapeutic test for syphilis, cautiously applied, by resolving the periaortitis perhaps, and freeing the aneurysm, made possible the revision of a diagnosis of tumor within six weeks in every one of 6 out of 19 cases in which this question was raised.

The electrocardiogram, while it cannot demonstrate the syphilitic character of a cardiac lesion, has great value in the determination of the extent of myocardial damage. It becomes therefore an essential to correct prognostic and treatment decisions.

The treatment of cardiovascular syphilis involves four problems: First, can the progress of an early case be arrested and by what measures? Second, how avoid therapeutic shock? Third, how interpret and control the so-called therapeutic paradox in both diagnosis and treatment, in which we clinically observe the progression of signs or symptoms with the supposed healing of the lesion? The fourth issue involves the differentiation, so far as is clinically possible, of two therapeutic groups—an intrinsically unfavorable type with pronounced coronary sclerosis and myocardial degeneration, and a favorable type with lesions limited largely to the aorta and relatively less involvement of the coronaries and myocardium.

The question as to whether early aortic syphilis can be completely arrested or not, cannot be fully answered as yet; but I would place my opinion on the affirmative side provided a proper selection and a sufficiently intensive treatment régime be employed. At best there will be failures. In the treatment of such cases, as well as in the therapeutic test to determine their syphilitic character, we witness the paradoxical progression in signs up to a certain point, coincident with healing of the lesion as determined by subsequent observation. The patient who at the outset of treatment had no more than a faint systolic murmur and a tambour second sound, will, before his lesion comes to arrest, have developed perhaps a slight hypertension, a definite additional diastolic murmur, evidencing a regurgitation from valve shrinkage, and slight signs of myocardial distress and cardiac dilatation from which he recovers as compensation is established. In fact, the very appearance of these signs under treatment is part of the positive therapeutic test. His myocardium will bear up under the slight fibrosis; his coronaries will remain patent in the face of some fibrous constriction in the sinuses of Valsalva and along their course. Intensive treatment with bismuth, mercury and iodides, followed after several weeks or months' preparation by the systematic prolonged use of arsphenamin in alternation and combination, offers, I believe, the best outlook.

But it is far otherwise in the case of the coronary sclerotic, with angina pectoris and a failing myocardium. To initiate treatment with, or even to use at all, drugs of the arsphenamin group in patients of this type too often invites sudden disaster, as a local reaction in an active lesion closes a coronary orifice or shrinks the circulation of an already laboring myocardium. Quite as disconcerting is the patient whose transient but marked symptomatic relief at the outset is transformed after a few weeks or months into a return of anginal attacks with increasing severity and the electrocardiographic and clinical signs of an irremediably damaged heart muscle. The cautious practitioner in treating all late syphilis seeks full investigation of the syphilitic's heart. The first clue to the situation may be furnished by an irreversible blood Wassermann test. He gives great weight to coronary symptoms. He prepares his cardiovascular patient in every case with small doses of bismuth or with mercury by inunction or, in a threatening lesion, with a soluble mercurial salt intramuscularly if the kidney permits. He

uses neoarsphenamin and sulpharsphenamin much more often than arsphenamin, in very small but ascending doses, in long series, with the patient at rest. In the badly damaged patient he avoids them entirely perhaps. In the young and hopeful case he pushes them vigorously after a few weeks of preparation, in the hope of securing complete arrest. What of aneurysm? Do not use arsphenamin at the start, for rupture may follow within a few hours from therapeutic shock. After a reasonably prolonged preparation, arsphenamin offers the small or moderate-sized aneurysm most gratifying prospects of symptomatic relief, prolonged arrest and even reduction in the size of the lesion. In all cardiovascular syphilis the intelligent treatment of the heart as a heart, and of the patient as a patient, runs parallel with but does not outweigh, as some cardiologists seem to feel, the treatment of syphilis as syphilis.

I have selected as my contribution to the discussion of visceral involvement some remarks on the gastrointestinal tract and the liver and spleen, constituting the upper abdomen of syphilis.

Stomach symptoms in syphilis, and syphilis of the stomach, are two different things. The former are common, being essentially accompaniments of that ubiquitous presence, syphilis of the nervous system, and its fellow, syphilis of the heart. Syphilis of the stomach, apart from the rather common subacidity of the young syphilitic patient, is a comparative rarity. It follows, therefore, that the syphilitic who has stomach trouble should be examined for syphilis of the nervous system and heart, and treated effectively for it if it is found. Merely to give him a "shot" or two of arsphenamin, because he has syphilis, is unpardonable. The fact that 75 per cent of syphilitic patients with stomach trouble have neurosyphilis and only 8 per cent syphilis of the stomach, points the moral that a spinal-fluid examination is part of the study of stomach symptoms in a patient with syphilis; and that even if it be negative, the gastric crisis, so often accompanied by a negative blood and spinal fluid, should be considered in a painstaking search for signs of tabes. Even though the patient have a duodenal ulcer, demonstrable by *x*-ray, and with a perfect symptomatic picture, the spinal fluid often reveals a neurosyphilis, and the symptoms, though not the signs, disappear with treatment for syphilis—a false therapeutic test. The surgeon is the conspicuous offender in dealing with syphilitic stomach trouble. He errs largely in a disposition to lean

upon the patient's story rather than the results of objective examination, to neglect or ignore the positive Wassermann test, and to prefer the dashing decision of exploration to the slower but more convincing method of therapeutic test, even when no *bona fide* emergency such as perforation of an ulcer, operable malignancy or question of an acute inflammatory lesion exists. Syphilitic gastric or round ulcer, which unquestionably exists, is so promptly transferred from the patient to the specimen bottle in the ever popular gastroenterostomy, that the clinical demonstration of its ready curability by antisyphilitic treatment is rendered artificially difficult. True gastric syphilids are so rare that they take little place here. Few aspects of late syphilis respond so brilliantly to the immediate application of arsphenamin treatment or reach a better ultimate result. In the vexing problem of differentiating syphilitic from carcinomatous lesions of the stomach, recall that syphilis as well as carcinoma is a notorious producer of achlorhydria. Evidence of bleeding seems to be worth more as a sign of carcinoma, for syphilitic ulcers everywhere are definitely not inclined to bleed. No time should be lost in therapeutic tests for syphilis of the stomach, even though the blood Wassermann reaction be positive, if roentgenologic examination shows an operable lesion and the clinical background suggests carcinoma. If an exploration is done and a syphilid found, the surgeon should not be in too great haste to do wholesale gastrectomy unless there is a high degree of obstruction from cirrhotic contracture. The response of gastric syphilids to treatment is often better than the results of extensive surgery, plus treatment as an afterthought.

The rectum as a site for supposed syphilitic lesions has held, I believe, a place of undeserved prominence in the estimation of clinicians. Syphilis of the rectum, and particularly rectal stricture of syphilitic origin, is not easy of convincing demonstration. Less than 22 per cent of 258 rectal strictures examined by Buie in the Mayo Clinic were associated with syphilis. More than half were the sequels of various forms of trauma, and 20 per cent were associated with nonspecific ulcerative colitis. It is an unusual thing to secure a convincing therapeutic test in stricture of the rectum, for parasitic enterocolitis and proctitis, as well as occasional cases of chronic ulcerative colitis, improve under arsphenamin treatment.

Syphilis of the liver and spleen is a phase of visceral syphilis of unusual interest to diagnostician and therapist. In no aspect

of the disease does the mimicry for which syphilis is famous reach a greater perfection, nor the patient sustain more disadvantage or the physician and surgeon more discomfiture thereby. McCrae has ably pointed out the disparity between postmortem and clinical findings. In my own experience, using the single criterion of palpable enlargement as evidence of abnormality, 15 per cent of more than 400 patients with late syphilis, excluding those with indubitable syphilitic hepatitis and splenitis, had what might be called the "latent" liver or spleen. The detection of hepatosplenic syphilis at this stage might well reduce the late and serious aspects to an almost negligible incidence. Wassermann positiveness in hepatosplenic syphilis seems to be higher than in almost any other late phase of the disease, with only 10 per cent of undoubted seronegative hepatic syphilis in my personal observation. The syphilologist's preventive effort, therefore, must be devoted to a strenuous advocacy of the routine blood Wassermann test in all medical examinations and particularly toward inducing examiners to take seriously and properly study any enlargement of either liver or spleen even in well persons. There is no difficulty at all in collecting repeated illustrations of the way in which a routine blood Wassermann test before surgical exploration would have saved both patient and surgeon risk and vexation.

The syphilitic hepatosplenic complex, like the cardiovascular group, involves two symptomatic types—the one associated with gumma and perihepatitis, the other with cirrhotic changes. The latter is relatively familiar through the decades of emphasis which have been placed upon hepatic cirrhosis. The former seems, for some reason, to have been less fully appreciated and to be a source of greater diagnostic confusion. Distention, stomach trouble and hemorrhage stand out in the story of the cirrhotic. But jaundice, attacks of pain, nausea, vomiting, chills and fever, the syndrome associated more especially with perihepatitis and gumma, seems insistently to deflect attention to the gall-bladder and to lead to a needless exploration. There seems not to be a symptom or combination of symptoms associated with nonspecific gall-bladder disease that syphilis of the liver and syphilitic perihepatitis cannot reproduce. Rolleston, even in the pre-Wassermann days, wisely placed mistaken diagnoses of cholelithiasis among the first of the errors of diagnosis in syphilis of the liver.

Of the causes of incorrect diagnosis, failure properly to use

the blood Wassermann reaction, as I have said, stands first. Next in rank stands unguarded reliance on the physical findings elicited by palpation of the abdomen. Sixty per cent of our mistakes were the product of the belief that there is anything about the position and characteristics of a mass in the upper abdomen that eliminates syphilis from the diagnosis or relieves the examiner of the necessity of reckoning with it. Next in rank come supposed gall-bladder symptoms, then supposed renal symptoms, then suspected malignancy, and following a whole group of minor items ranging from hepatic ptosis, through angina pectoris and pancreatic cyst. Again and again the value of the blood Wassermann test as a predecessor to all abdominal operations, and of a therapeutic test in gall-bladder syndromes in patients with syphilis for whom operation is not urgent, comes to the front.

Time prevents my discussing the interesting problem of the interpretation of jaundice in the syphilitic patient. I can only urge that not all jaundice in patients under treatment with arsphenamin is due to the drug—in fact, only a small part of it probably is. The relapse of the inadequately treated syphilitic liver (hepatorecurrence of Milian), the progress of an hepatitis under inadequate treatment, acute yellow atrophy of syphilitic or nonsyphilitic origin, intercurrent infectious and catarrhal jaundice all deserve consideration side by side with arsenical toxic effects and the surgical causes of jaundice, in analyzing any given case. The clinician must remain open-minded toward all the possibilities to achieve the highest accuracy in diagnosis.

The treatment of early syphilitic hepatitis in the so-called "latent liver" stage does not differ materially from that of early or latent syphilis. Arsphenamin, mercury, bismuth, iodides may all be vigorously employed. It is wise to begin with iodide and mercury or with bismuth in the later cases at this stage, and to persist in combined treatment for a long time to control the serious tendency to relapse. The therapeutic problem of advanced syphilis of the liver is not unlike that of the badly damaged heart. The disastrous effect of sudden and vigorous application of the arsphenamins, which induces a therapeutic paradox, must be apparent to anyone of experience. Theoretically, if it were possible to have localized gumma in pure form or accurately differentiated from cirrhotic changes, it would be possible to secure excellent results in not too extensive gummatous cases by arsphenamin used at the outset.

Only in this way can one explain the really brilliant outcomes sometimes observed after arsphenamin before the limitations of the method were understood. But arsphenamin is almost fatal to the cirrhotic, for it simply increases the fibrosis which is the chief pathological cause of his difficulties. It follows, therefore, that the patient with a syphilitic liver whose symptoms and findings, including ascites, suggest an extensive cirrhosis, is not a candidate for arsphenamin, but for mercury and iodides over a long period. Personally I feel that properly selected cases of this type, after months of preparation, and sometimes immediately (as with severe anemias), may be advantaged by the skilful and conservative use of small doses of neoarsphenamin. But for the general practitioner, and in fact for the expert too, the differentiation of a gummatous from a cirrhotic syndrome is too precarious and uncertain to justify much therapeutic confidence. It is wiser to treat late syphilis of the liver from the outset with mercury and iodides, or perhaps bismuth, rather than arsphenamin. Ascites always introduces a serious aspect, and the outlook for an excellent result in the patient who has progressed to this point is only one-sixth as good as that of the patient without ascites. In the management of this complication, tapping, too often the first resort, seems to me greatly inferior to reduction of the fluid by limitation of intake, catharsis and diuresis, notwithstanding the occasional surprises of a spontaneous or traumatic Talma-Morison effect following repeated tappings, as reported by Riesman. The successful treatment of hepatic syphilis is a matter for years, not weeks, and results should not be too hastily compiled and labelled bad or good. Some of my apparently best cases have gone the road of hemorrhage from varices, while others, seemingly much worse, are now about their usual work.

Of the spleen in syphilis it may be said therapeutically that it stands treatment better and yields more surprises than the liver. Obstinately unresponsive at times, it will in the most surprising fashion yield to moderate or intensive treatment while the surgeon is scrubbing up for the splenectomy. Of splenectomy on the syphilitic spleen, I can only say that it is a formidable operation with risks often much greater than those of prolonged treatment for syphilis. I feel that in general it has been too early invoked, and that only after two years instead of two months of arsphenamin, iodid and mercury is it justifiable to urge the operation for a persistent asthenia and anemia, provided no other cause than the syphilis can be found. Even then the miracle may fail to appear.

I speak in closing of syphilis of the lung and mediastinum, only to urge the possibility of expanding this field to the great advantage of the patient, by the intelligent use of the therapeutic test so well advocated by Fitz-Hugh. In such tests, the nonspecific effects of arsphenamin must never be forgotten, for pulmonary spirochetosis may be cured and even pulmonary tuberculosis may react favorably to this drug. Such tests should be performed with mercury and iodid, pending a precise determination of the effect of bismuth, which will probably resemble that of arsphenamin. To witness the cure of an extensive mediastinopericarditis, of supposed pulmonary malignancy with metastasis to the other lung, of the acute pulmonary lesions and unresolved pneumonias described by our Philadelphia confrères, under treatment for syphilis, presages a revision of our conceptions of the frequency and importance of what has hitherto been regarded as an untenanted rather than merely a misunderstood field in clinical syphilology.

THE TREATMENT OF CENTRAL NERVOUS SYSTEM SYPHILIS*

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It is difficult to say anything about the treatment of central nervous system syphilis which has not already been authoritatively discussed by Fordyce and by Stokes and Shaffer. These workers, in 1924, each published papers, the one describing treatment methods and results in 405 cases, and the other in 443 cases of neurosyphilis. Their results (except for certain data as to paresis) are the most complete available in the literature and should be familiar to everyone who attempts to treat neurosyphilis.

In a discussion limited to a few moments it seems wise briefly to outline the methods of treatment in use in the Syphilis Clinic of the Johns Hopkins Hospital and to present a summary of the results obtained in different types of neurosyphilis.

The phrase "types of neurosyphilis" touches at once upon the most important aspect of the treatment question. It is of the utmost importance to the patient that his disease be correctly classified before treatment is begun. The experience of the past ten years has taught all of us that what suffices to restore one patient to physical and serologic normality makes no impression on another. The treatment of tabetic optic atrophy is quite different from that of a neurorecurrence. As Bunker has aptly put it: "It is not a question of the diagnosis of syphilis in a given patient, nor even of the diagnosis of neurosyphilis itself; our problem deals only with the patient in whom syphilitic involvement of the nervous system is known to be present, the question being: What then is the proper treatment for that patient?"

A study of this question must be made for each individual patient, since in no phase of syphilitic infection is extreme individualization

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of treatment more desirable than in neurosyphilis. It involves, to a certain extent, the cataloguing of each patient into a diagnostic subgroup, a corresponding cataloguing of treatment methods, and an attempt to fit the appropriate group of treatment procedures to the appropriate case.

One may subdivide the clinical manifestations of neurosyphilis into many groups, or, much more simply and appropriately for a general survey of the field, adopt the classification proposed in its major elements by Head and Fearnsides, and more compactly still by Stokes and Shaffer. This divides all cases, chiefly on the basis of pathological anatomy, into three main groups:

1. Purely meningeal neurosyphilis. This is almost always an early manifestation, within the first two years of the infection. In our own material it includes acute syphilitic meningitis, neurorecurrences and early asymptomatic neurosyphilis.

2. Neurosyphilis with predominant meningeal or vascular involvement or both, sometimes with and sometimes without parenchymatous changes. If present, these latter are minimal and rarely enter into the clinical picture. This group is the general catch-basket. It includes diffuse cerebrospinal syphilis and such widely separated entities as brain gumma, syphilitic hemiplegia, combined system disease and late asymptomatic neurosyphilis.

3. Predominantly parenchymatous neurosyphilis, including tabes and paresis.

For practical purposes only, both Fordyce and we have added a fourth group—the syphilitic optic atrophies.

We are accustomed to tell students that at present we have available, subject to individual modifications, five treatment systems for neurosyphilis, which may be briefly summarized as follows:

1. Routine treatment, consisting of courses of an arsphenamin alternating with courses of mercury or bismuth, together with potassium iodide by mouth. A representative arsphenamin course is six to eight weekly injections; a representative interim course, two to four months. Except in unusual circumstances, arsphenamin or silver-arsphenamin is the drug of choice; neoarsphenamin rarely and only for extraneous complicating factors; sulpharsphenamin not at all, in spite of its preliminary puffs in the literature. Our use of sulpharsphenamin produced an outcrop of serious reactions, and we could see no advantage in results over other arsphenamin products.

This arrangement of alternating routine treatment is essentially that in use for patients without nervous system involvement. We employ it only when the evidence of neurosyphilis is minimal, that is, chiefly in early or late syphilis with minor spinal fluid changes or in late syphilis with minor neurologic signs and a negative spinal fluid.

2. This is an intensification of the routine treatment in the direction of more arsphenamin—larger doses, longer courses, shorter intervals between doses. The average course is twelve to sixteen injections of arsphenamin or silver-arsphenamin, usually the former. Here, as in the routine system, treatment is usually continuous, though, except in the case of early meningeal neurosyphilis, we do not regard a rest period as an almost irreparable calamity, as it may be in primary or secondary syphilis. Individualization is here not only permissible but essential. It will be noted that since we deal for the most part with ambulatory rather than with hospitalized patients, we cannot carry out such intensive treatment as is recommended by Stokes and Shaffer, including daily intramuscular soluble mercurials and intravenous sodium iodide.

This treatment system is used in practically all cases of early meningeal neurosyphilis, with whom it is rarely necessary to resort to still more intensive measures, and in the majority of patients with diffuse meningovascular neurosyphilis.

3. The addition to the treatment scheme of intraspinal treatment by the original Swift-Ellis technic, with only minor modifications. We have tried and abandoned as too risky most of the proposed alterations, such as Ogilvie's method of reinforcing the serum or its administration by the intracisternal or intraventricular route. Lower-cord reactions have been practically eliminated by spacing treatments two weeks apart, giving six or less to a series and avoiding the injection of more than 20 cc. of serum at one treatment. Fordyce's technic of injection is used.

In our experience, intraspinal treatment has been chiefly valuable in two restricted groups of cases: in tabes, especially those patients with lightning pains, sphincter disturbances and ataxia, and in optic atrophy, whether tabetic or not in origin. These points will be more fully discussed presently. We do not turn now, as we once did, to intraspinal treatment for serologically resistent cases of meningovascular neurosyphilis. Better results are, we believe, obtained by one or both of the next systems to be mentioned.

4. The fourth treatment system involves the prolonged use of tryparsamide, either with mercury or bismuth alone, or in combination with an arsphenamin. No detailed description of our method of use of this drug can be attempted, since full reports have recently been published.

Tryparsamide is of particular value in paresis, asymptomatic neurosyphilis with Group III (paretic formula) spinal fluid; also in certain cases of tabes or diffuse meningovascular neurosyphilis with spinal fluid changes of the paretic type.

5. The infectious disease method of treatment—malaria, relapsing fever and, more recently, rat-bite fever. Although we began the use of malaria about two years ago, no patients so treated are included in the results presently to be mentioned. Until very recently the use of the infectious disease treatment has been confined to paresis; reports of its results in tabes, optic atrophy, etc., are now beginning to appear. Its worth in paresis appears to be firmly established. In a series of more than 1200 cases which I have compiled from the literature, complete remissions and restoration to normal activity were brought about in 31.4 per cent. Our use of this form of treatment is not limited to paresis. We are applying it also to patients serologically resistant to other treatment schemes, especially to tryparsamide.

I have been able to gather the records of 203 neurosyphilitic patients followed for an average period of four years. Of these 34 had early meningeal neurosyphilis, 60 late diffuse meningovascular involvement, 57 tabes, 35 paresis and 17 optic atrophy, tabetic and otherwise.

I have attempted the same sort of correlation between clinical and serologic results as that of Stokes and Shaffer, and an estimate of combined results in terms of *excellent*, *good*, *fair* and *poor*. The term excellent is applied only to those cases who were freed of symptoms or were left with only trifling residuals and rendered serologically negative in blood and spinal fluid, with this condition maintained for from one to thirteen years. To conserve time and to avoid tiresome statistical repetition, only the percentage incidence of excellent results will be considered.

Of 34 patients with early meningeal neurosyphilis, 67.6 per cent obtained an excellent result after an average of twenty months treatment. This compares unfavorably with Fordyce's 100 per cent and Stokes' 90.9 per cent. The difference is probably one of

classification, since 13 of our 34 patients were Group III asymptomatic neurosyphilis and might be regarded as *paresis sine paresi*. Three such individuals, with a result classified as poor, have subsequently developed frank clinical neurosyphilis.

Almost exactly the same proportion—66 per cent—of the patients with late meningovascular neurosyphilis had an excellent result from an average of two years' treatment. This is as compared with 84.6 per cent of Fordyce's series and 74 per cent of Stokes' cases. Here again the figures are not quite comparable, due to the inclusion in our series of a number of serologic preparetics. Four patients of this group have subsequently developed paresis.

Twenty-one of the 94 patients in these two groups were treated intraspinally. The end-results were no better than when intraspinal treatment was omitted, though in certain patients treatment was appreciably shortened by its use. This fact, together with the eminently satisfactory results obtained from tryparsamide, have led us practically to abandon intraspinal treatment in these types of neurosyphilis.

The situation, however, is quite different in tabes. The 57 tabetics are almost equally divided into two groups. Twenty-eight were treated intraspinally, 29 were not. Of the former group 46.4 per cent had an excellent result. Of the latter only 20.6 per cent—less than half as many—were equally fortunate. Moreover, the length of time under treatment was perceptibly shortened when the intraspinal route was used. Our results from intraspinal treatment in tabes are almost identical with the 50 per cent excellent result reported by Fordyce and the 48 per cent by Stokes. Of the patients classified as poor results, many had either gastric crises or Charcot joints, lesions notoriously resistant to treatment.

As to paresis, our results are differently expressed. Here stress is laid on clinical remission without insistence on serologic normality. In a sense, also, our material is selected from a group of early paretics since we deal largely with ambulatory patients sufficiently well mentally to avoid sanitarium or hospital commitment. It is chiefly indicative of the value of tryparsamide in early paresis that we can report 20 complete remissions, or 57 per cent, among 35 paretic patients *not treated with malaria*. In 11 or these patients the remission has lasted three or more years (2 of seven years, 1 of nine years), and in 9 from one to three years. Fordyce also reports 54 per cent remissions in a series of 118 cases (remissions usually

short), and as good or better results have been obtained by Solomon. I do not agree with the attitude that paresis is a diagnosis permissible only after therapeutic failure, nor do I feel that its treatment is hopeless. When early recognition of the disease can permit 50 per cent remissions, often of years' duration, much has been accomplished even without malaria. The points of superiority of malaria, as apparent from the literature and our own experience, are that it offers a 30 per cent hope of remission even for the advanced case, and remissions are apparently longer than heretofore.

The survey of our whole material has offered strong evidence of the serious prognostic import of the paretic formula in the spinal fluid. Separating our tabetics into two groups—those who showed Group III fluids and those who did not—only 19 per cent of the former obtained an excellent therapeutic result as against 43 per cent of the latter. For early meningeal neurosyphilis the corresponding figures are 38 and 82 per cent, and for the late diffuse meningovascular type, 56 and 79 per cent. These differences are too uniform and too striking to be without significance. I interpret them to mean that a neurosyphilitic patient with a paretic fluid is an ultimate candidate for tryparsamide, malaria or both, though not necessarily for paresis. And in practice this is the method of attack which we are now adopting.

No class of neurosyphilitic patients is so tragic as the primary optic atrophies. Without here attempting an ophthalmologic classification of our material, or to differentiate tabetic atrophy from that due to basilar meningitis, I wish to emphasize what I believe to be an imporatnt point in treatment. I do not agree with certain ophthalmologists, or with Stokes, in the opinion that arsphenamin is contraindicated in primary optic atrophy. Given a patient with early but rapidly advancing optic atrophy, I am convinced that blindness can be prevented only in one way—by the prompt and energetic use of intravenous arsphenamin *plus intraspinal treatment,* the latter more important than the former. In 12 of 17 patients with syphilitic primary optic atrophy, this system of treatment arrested the rapid visual failure; in 3 visual acuity and fields were improved over the admission examination, and in 9 the process remained stationary. The serologic results were also excellent in 7 of these 12 patients. In a much larger series of cases treated with arsphenamin intravenously *without* intraspinal treatment or with mercury, bismuth or potassium iodide, every patient has progressed

inexorably to blindness. To us intraspinal treatment offers the only avenue of hope for the unfortunate victim of optic atrophy and we feel confident of at least 50 per cent good results in early case. Our opinion is borne out by Fordyce's 55 per cent successes in 39 patients.

It goes without saying that all patients in the series have been repeatedly reëxamined from the physical and laboratory standpoints during and after treatment. Little has been said about the total duration of treatment. In general we have attempted to apply to neurosyphilis our standard for early syphilis one year of continuous treatment after the blood and spinal fluid have become and have remained completely negative. The average length of treatment in the whole series has been a little over two years. Thereafter observation is continued for a lifetime.

Finally, a sentence which actually belongs at the beginning of this paper: If routine spinal puncture could be properly carried out in every patient with early syphilis, and treatment altered to meet the exigency of positive findings, there would be little neurosyphilis to treat.

In summary, the adequate treatment of neurosyphilis depends upon a thorough study of the patient and the means at hand for his treatment. At our hands certain types of neurosyphilis call for certain treatment systems. These are:

1. For "minimal" neurosyphilis, routine antisyphilitic treatment.
2. For early meningeal and late meningovascular neurosyphilis, an intensification of routine treatment. For serologically resistant cases from these groups, tryparsamide, malaria or both.
3. For selected tabetics, intraspinal treatment.
4. For paresis, tryparsamide, malaria or both.
5. For optic atrophy, intraspinal treatment.

Treatment must always be individualized and must often proceed according to a system of trial and error.

The results in this series of 203 patients compare favorably with the larger series of Fordyce and of Stokes and Shaffer. The three series contain 1051 patients, a material sufficiently large to make the results statistically convincing and to shed a ray of optimism over a field of therapy where hopelessness is still too frequently encountered.

SYPHILIS AND MARRIAGE IN RELATION TO SPECIFIC THERAPY*

By JOHN A. KOLMER, M.D.

Some of the most difficult and perplexing question in relation to the specific treatment of syphilis arise in connection with the subject of marriage and parenthood. These do not refer to those cases of syphilis in either sex contracted and thoroughly treated before marriage, but rather to those in whom evidences of syphilis are discovered after marriage and especially after children have been born. Can the father have syphilis without infecting either mother or child? Can the mother have syphilis without infecting the father or the child? We are fairly certain of what should constitute the criteria of cure before marriage and parenthood may be safely sanctioned, but numerous cases arise in which a father is found to present evidences of syphilis after marriage of several years which immediately raises the question: Are the mother and children infected and thereby in need of specific medication, even though they do not show any definite clinical or serological evidences of the disease? What is the proper procedure when a mother is found syphilitic under similar circumstances? Should the husband be treated? Should the children? Are we in position at present to lay down broad and uniform rules for the management of such cases? What are the criteria by which decisions may be reached? No one experienced in the treatment of syphilis will deny the tremendous practical importance of these questions, involving as they may the domestic peace and happiness as well as the health and even the lives of innocent and unsuspecting individuals. It is not so much a question of *how* such cases should be managed from the standpoint of specific therapy, because this is along the lines of the treatment of syphilis in general, but rather the question of diagnosis and the criteria by which decisions are reached regarding whether or not the spouse and children require antisyphilitic medication.

* Read October 6, 1926.

Great advances have been made in our exact knowledge of conjugal and congenital syphilis during the past twenty years by means of the Wassermann test, and especially during the past ten years by careful and extensive clinical, sociological and serological studies employing improved methods, but there are still many and wide gaps in our information on these subjects. Percentages expressing the incidence of infection under this or that condition aid us in arriving at decisions, but in many cases it is after all a matter of expressing opinions with a full realization that only the future welfare of the individuals concerned can tell us if we are right or wrong.

Owing to the frequency with which syphilis is transmitted to the spouse and offspring, the subject of syphilis and marriage has very properly attracted a great deal of attention, and especially in relation to public health. Many statistical studies have been published which need not here be reviewed, as our present interests concern the matter of how much specific treatment should be given either the man or woman before marriage may be sanctioned, in order to be fairly sure that the spouse and offspring will not be infected. In a way, the subject resolves itself into a discussion of the criteria of cure, because the only safe time to marry is when there are good reasons for believing that the individual has been cured. Furthermore, our standards and criteria are more strict than those laid down by Fournier and other older syphilologists, because they had only clinical signs for their guidance, whereas we have the Wassermann test in addition, which has added very materially to our knowledge of conjugal and congenital syphilis during the past fifteen years.

I may divide the subject, therefore, into two parts, namely: (*a*) the question of syphilis before marriage, and (*b*) its discovery after marriage. Relative to the first, involving the marriage of a syphilitic I am stating here the rules by which my opinions are guided, these being in conformity with the practice of many leading and experienced syphilologists:

1. Patients seen and placed upon intensive treatment during the chancre stage and before the Wassermann reaction has become positive may be allowed to marry after at least one year of vigorous treatment, followed by a second year of observation and milder treatment, provided there have been at no time secondary manifestations or serological evidences of infection in the blood and spinal fluid.

2. Patients seen and placed upon intensive treatment during the

late primary stage or in the secondary stage after the Wassermann reaction has become positive may be allowed to marry after two years of vigorous treatment, followed by a third year of milder treatment and observation, during which there have been no clinical manifestations of the disease, repeatedly negative blood Wassermann reactions, and at least one complete examination of the spinal fluid with negative results.

3. Patients seen in the latent and tertiary stages of syphilis with positive Wassermann reactions may be allowed to marry after thorough courses of treatment over a period of at least two years, followed by a third year of milder treatment and observation, during which there should be absolutely no clinical evidences of syphilis, repeatedly negative blood Wassermann reactions, and at least one complete examination of the spinal fluid with negative results. A provocative Wassermann test is advisable.

4. A man with a persistently positive blood Wassermann reaction may be allowed to marry if there has been at least three years of vigorous treatment and if the spinal fluid shows no abnormalities; in the majority of such instances, both the spouse and children escape infection, *but it cannot be stated that the union is absolutely free of danger of transmission of infection*, and many physicians, including myself, naturally hesitate to sanction marriage under these conditions. A woman under these conditions, however, should not marry at all, because she incurs a much greater risk of transmitting the disease to offspring; there is likewise a slight risk of transmission to the spouse.

5. A man or woman with a persistently positive blood Wassermann reaction and the spinal fluid changes of neurosyphilis should not marry, regardless of the amount of specific treatment received, because under these conditions there is more risk of transmission to both spouse and children.

The above-mentioned conditions are definite, and probably most physicians will agree in whole or most part with the conditions laid down in relation to marriage, but there is a larger group of more puzzling cases in which an opinion must be very guarded because of the impossibility in some cases of detecting latent syphilis by the most thorough physical, serological and roentgenological examinations.

6. If the patient is certain of having contracted syphilis several years previously, followed by a period of specific treatment, I

believe that marriage may be sanctioned if a thorough physical examination reveals no evidences of the disease and the blood and spinal-fluid reactions are negative. I refuse an opinion unless a spinal-fluid examination is permitted, and I believe that a provocative Wassermann test is advisable. If the latter is conducted, the spinal-fluid examination should be made when the provocative blood tests are conducted, in order to obtain the value of provocative spinal-fluid reactions, if such occur. If in spite of negative and favorable results of these kinds the patient and the physician wish to be on surer ground, a year or two of treatment may be given during which repeated physical and serological examinations may be made. In this case the year of specific treatment replaces the provocative injections of arsphenamine or neoarsphenamine, and it may be fashioned along the lines employed for the treatment of asymptomatic late syphilis.

7. If the patient, an old syphilitic, is unable to give a definite and clear account of his infection and of his previous treatment, greater caution is required. Likewise in cases of either sex with indefinite but suggestive venereal histories, a woman with a history of miscarriages, etc., a thorough physical examination should be made along with blood Wassermann and complete spinal-fluid examinations. A provocative Wassermann test is advisable and I always advise one probationary year during which at least two physical and four blood Wassermann tests are made; it is not usually necessary or advisable, however, to repeat the spinal-fluid examinations. If these examinations fail to disclose evidences of the disease, marriage may be sanctioned, but it is necessary to employ a sensitive complement-fixation test because of the danger of falsely negative reactions in the presence of extremely latent syphilis.

It scarcely requires stating that a syphilitic of either sex with open lesions, and especially chancres and mucous patches, runs grave risks of infecting the sexual partner and especially by repeated contacts. In addition there are risks of extra-genital infection, and marriage at least can never be sanctioned under such circumstances.

But in the presence of clinically latent syphilis without demonstrable open lesions, what are the chances of infection of the spouse? The subject must be discussed separately according to the sex of the syphilitic partner.

A syphilitic husband under these conditions is far more likely to infect his wife than is a syphilitic wife likely to infect her husband.

She runs the risk of infection by three ways, namely: contact with an open lesion of the husband, which may develop at any time; contact with his semen, carrying *Sp. pallida;* and third, the possibility of infection from a syphilized fetus, if it is granted that paternal conceptional syphilis may occur. The husband, however, is liable to infection from a syphilitic wife only in case he comes in contact with open lesions, if such develop in the course of years.

Infection of a wife by contact with the open lesions of a husband requires no discussion; if one can be certain that the disease has been latent through the period of marital relationship, infection of the wife by this route may be excluded. But I believe she may be infected by the semen direct without involving the question of pregnancy, and especially if her husband's testicles are infected with *Sp. pallida*, as is now regarded of frequent occurrence even in the absence of clinical signs of infection. In such cases the disease may run a very mild course without its presence being suspected, the wife being entirely ignorant of infection (*syphilis ignota*), because the spirochetes are of greatly reduced virulence.

But is it possible for a woman to carry a syphilitic fetus and yet escape the disease? Some physicians believe that *Sp. pallida*, or rather a smaller sporelike form of the parasite, may infect the ovum direct (paternal conceptional syphilis); but may the mother escape through the destruction of spirochetes by placental ferments, as stated by Routh or some other means? Jeans has found that 85 per cent of mothers of syphilitic infants gave positive Wassermann reactions. But what is the status of those who do not and who present no clinical evidences of the disease? For my own part I believe that we should regard all mothers of syphilitic children born at or near term as syphilitic, and place them on antisyphilitic treatment. It may be that a woman aborting a syphilitic fetus, before the third month, may escape infection, but I doubt this and feel absolutely sure that all women with a history of two or more syphilitic pregnancies should be placed on antisyphilitic treatment. Indeed, an increasing number of syphilologists believe that paternal infection of the ovum does not occur and that a syphilitic fetus always indicates that the mother is syphilitic and that she was infected before conception occurred. In other words, according to this view, a syphilitic fetus always indicates syphilitic infection of the mother, and I subscribe to this opinion, at least to the extent of always advising antisyphilitic treatment of the mother even though her

blood Wassermann test may yield a negative reaction. Indeed, I know of no other type of syphilis more likely to yield *falsely negative* Wassermann reactions unless it be latent congenital syphilis of children.

However, it must be granted that a woman married to a man infected with syphilis five or more years previously may escape seminal and fetal infection if her husband's disease has been latent. But our opinion in each individual case must be based only upon thorough physical and serological examinations, because if the wife is syphilitic, the disease is apt to be very latent and defy detection even by thorough and sensitive tests. However, we must have some criteria for judging whether or not the wife of a syphilitic man requires antisyphilitic treatment, and I believe that treatment may be omitted under the following conditions: (*a*) When there are no manifest or suspicious clinical evidences of syphilis; (*b*) when repeated Wassermann tests by a sensitive and acceptable method yield negative reactions; (*c*) when there is no history of spontaneous abortions or miscarriages; (*d*) when the child or children show no clinical or serological evidences of syphilis. To these may be added negative provocative blood Wassermann and spinal-fluid reactions, although these cannot usually be applied when conditions demand or warrant the secret investigation of spouse and offspring.

But if these are acceptable as criteria for the omission of specific medication for the wife of a syphilitic man, what are the indications for the institution of treatment? I believe these may be stated as follows: (*a*) The presence of even suspicious clinical manifestations even though Wassermann reactions are negative; (*b*) the occurrence of a positive Wassermann reaction, even though there are no clinical manifestations; (*c*) the birth of a syphilitic fetus or child even though there are neither clinical manifestations nor positive Wassermann reactions in the mother. If the blood Wassermann reactions are negative but the spinal fluid shows abnormalities, treatment should be instituted.

As a general rule, most reliance is placed upon the results of Wassermann tests, and especially since these are frequently the best examinations possible when conditions demand an investigation without disclosing its purpose. As repeatedly stated, it is imperative that these tests shall be done with a method as sensitive as consistent with specificity because even under these circumstances we are always apt to secure falsely negative reactions. After a

woman has lived with a syphilitic man for several years we can never be absolutely sure of her having escaped infection; for example, as shown by Veeder, at least 86 per cent of mothers of syphilitic children yield positive Wassermann reactions indicating syphilitic infection even though there are no demonstrable clinical evidences and the women are ignorant of being infected (*syphilis ignota*). At best we may say that in the absence of all manifest or suspicious clinical and serological evidences of infection antisyphilitic treatment need not be given, but that there should be subsequent and periodic clinical and serological examinations over a period of several years. As a general rule these may be managed even when it is necessary to proceed secretly in order to preserve domestic peace and happiness. But no physician should be a party to any agreement by which the spouse is neglected and left to her fate; with tact and good judgment the physician may readily do his duty even in difficult situations when suspicion has greatly sharpened the intellect of the wife.

The reversal of these conditions may present just as puzzling a situation—that is, when syphilis is brought into a marital relationship by the woman. If she presents early open lesions it is almost sure that the husband will become infected with the production of a chancre and the usual clinical course of syphilis. But in the absence of these, what are the chances of infection of the husband when the disease in the wife is chronic and latent with no demonstrable open lesions? The possibilities of his contracting the disease are very much less than when the conditions are reversed and the syphilitic partner is the male. But yet there is danger of infection, and especially after marriage of several years. Evidently small open lesions may develop about the woman which do not attract attention, and, curiously enough, the man may not notice a chancre or other manifestations of sufficient degree to attract attention, the presence of the disease being unsuspected because he is ignorant of the possibilities of infection. Numerous instances are on record of neurosyphilis in the husband contracted from marriage with a neurosyphilitic woman, although in most cases the reverse pertains. Evidently the spirochetes derived from small undetected lesions about the woman are of greatly reduced virulence and produce undetected lesions at the point of entry. Therefore, if syphilis has been introduced into a marriage by the wife, the husband should be examined for evidences of infection, and when this must be secretly

done I know of no more difficult situation. As a general rule, it is possible to arrange for a physical examination and a blood Wassermann test at least, and if the latter is positive by a sensitive and acceptable method, it may be assumed that the husband is infected and in need of specific treatment. If these examinations are negative a provocative Wassermann test and examination of the spinal fluid should be done if possible; if these are negative we may at least state that there are no indications for antisyphilitic treatment, although we may not be absolutely sure that latent syphilitic infection is absent.

When syphilis is discovered in either spouse under such conditions, it is almost impossible to give thorough treatment without disclosing its purpose; at least, in my experience, it is difficult to secure proper coöperation until the patient understands the purpose of treatment. I have in many cases, however, given courses of neoarsphenamine by intravenous injection on the assumption that these were required for the correction of anemic states, and have succeeded in administering mercury by inunction in the form of 85-per-cent calomel ointment under the disguise of massage. Mercury and iodide at least may be easily given by ingestion over long periods of time after Fournier's chronic intermittent method. But if possible, the plan of therapeutic attack should be along the same lines as advised for the treatment of chronic syphilis according to the condition of the spouse, although modifications are usually demanded by the individual conditions and circumstances of each patient.

THE SYMPTOMATOLOGY AND TREATMENT OF CONGENITAL SYPHILIS*

By JAY FRANK SCHAMBERG, M.D.

Within the quarter-hour allotted for this presentation one can merely paint the picture of congenital syphilis in crude and bold brush-strokes. The term "congenital syphilis" is here used advisedly to avoid the designation hereditary or heredosyphilis, which, though thoroughly ingrained in medical literature, is from a biologic standpoint an unscientific misnomer. There is scarcely more reason to speak of heredosyphilis than there is to use the term heredosmallpox, for children are at times born with a variolous eruption or with the scars resulting therefrom. Before the days of the Wassermann test our knowledge of the symptomatology of syphilis transmitted from parent to child was limited to the secondary eruptions which appeared at or shortly after birth, to later tertiary cutaneous lesions, to ocular and auditory disease, dental and osseous abnormalities, and to visceral and nervous diseases. I shall speak of these symptoms with relative brevity, for they have been known for a half-century or more. Perhaps of greater interest will be the subtle and poorly defined evidences, with or without a positive complement-fixation test, in the children of luetic parentage.

There is no clinical primary lesion in congenital lues unless the syphilis is contracted during the delivery of the infant. The early symptoms of congenital lues are comparable with the secondary period of acquired syphilis in adults. To be sure, some of these symptoms are never seen in adult syphilis. Manifestations may be present at birth—particularly the senile facies, bad nutrition, sometimes going on to a marasmic state, hoarse cry, snuffles, yellow skin, enlarged liver and spleen, and certain skeletal defects. The most common eruptions are the bullous type, particularly involving the palms and soles, and the macular syphilides affecting the diaper region and looking somewhat like an eczema. Not infrequently one observes a glazed, dull-red appearance of the palmar and plantar

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skin. Other eruptions may be papular, crusted or ulcerative; not uncommonly ringed-shaped erythematous patches are present. The mouth, anus and vulva may exhibit mucous patches. Fissures or rhagades about the lips and nose are highly characteristic and often leave indelible scars.

The symptoms referred to are more prone to develop some weeks after birth than to be present at the time of delivery. In about two-thirds of the cases they appear in from three to six or eight weeks. The Wassermann reaction is positive in the vast majority of these cases, and the diagnosis is as a rule not difficult.

Many congenital luetics entirely escape secondary symptoms. Of greater interest are the late manifestations of congenital syphilis, called by Fournier "syphilis hereditaire tardive." These are comparable with tertiary adult syphilis and, as may readily be surmised, may involve any tissue or organ. One of the most common presenting symptoms is interstitial keratitis, which occurs in a considerable proportion of cases of late congenital lues. Chorioretinitis is the next most frequent ocular manifestation. In many of these cases the so-called Hutchinson teeth are also present. The test teeth are the upper central incisors — in classic cases they are crescentically notched, peg-shaped and separated. There are various modifications of this condition, and there are also other dental abnormalities seen in congenital syphilitics which are of some evidential value. Hutchinson's triad of interstitial keratitis, characteristic teeth and eighth-nerve deafness is pathognomonic, but rarely seen in its completeness.

Linear scars radiating from the commissures of the mouth are highly suggestive, as are likewise the saber-blade tibia, hydrarthrosis of the knee-joints, osteitis, periostitis, dactylitis and epiphysitis. Marked frontal protuberances are suggestive, and the saddleback nose is a striking stigma. Paroxysmal hemoglobinuria is so often of syphilitic origin that its existence should always excite suspicion.

The nervous system involvement is represented by meningitis, epilepsy and various palsies, juvenile tabes and paresis.

The longer the period of time elapsing between the contraction of syphilis by the parent or parents and the birth of the infant, the more attenuated and ill-defined will be the symptoms of syphilis in the infant, if the disease is transmitted to it. To be sure, the amount of treatment of the parent or parents will also influence this. Thus there may be abortion or stillbirth, secondary symptoms at or

shortly after birth, late tertiary manifestations at the age of three, six, ten, twenty or thirty years, or there may never be a single stigma discoverable on the skin or elsewhere. There are numerous cases of congenital syphilis in which the sole symptom is a positive Wassermann. We have examined in our clinic large families of syphilitic parents and have at times found four out of five or five out of six children with positive Wassermanns and no other discoverable symptoms. What the ultimate fate of such children will be only careful studies and life-insurance statistics would determine. On the other hand, there are many cases of late congenital lues with clinical signs in which the Wassermann is negative. Indeed, the older the patient the more apt is this to be the case, for time is a distinct factor in the spontaneous extinction of the Wassermann reaction. Again, there are cases of children born of luetic parents in whom the Wassermann is negative and no definite stigmata of syphilis exist, but who present what has been aptly called a "constitutional inferiority." They are often anemic and underweight without other discoverable cause: they commonly lack endurance and are hypersusceptible to various infections. Their general health improves only under specific medication. Some of these persons as they grow to manhood and womanhood exhibit premature arterial fibrosis. This group of patients offers a most fertile field for future study.

In this connection, too, great interest attaches to infants born of syphilitic mothers, in whom the umbilical-cord blood is strongly positive at birth, but in whom the Wassermann becomes negative a week or ten days later. Fordyce and Rosen had under observation 140 such infants who were well after repeated examinations. They advise no treatment for such cases, but surveillance for several years.

Treatment. There is no crystallized formula for the treatment of syphilis; there are many formulæ. Most clinicians of experience have adhered to the method which has given them the best results. It takes time to effect a comparative evaluation of these methods. There may be differences in the relative choice of drugs, their combination, the routes of administration, the sequence of their use, the dosage and other details, but there is a broad underlying principle common to all. There must be an adequacy of treatment with effective agents; there must be persistent therapeutic effort irrespective of the outcome of the Wassermann; the treatment must be

aided by serologic tests of the blood and spinal fluid, and there must be subsequent periods of surveillance.

The most important treatment of congenital syphilis is *prevention*. This may be aided by employment of the Wassermann test on candidates for marriage, the thorough treatment of syphilitics and rigorous criteria of consent to marriage, the routine use of the Wassermann in pregnant women, and appropriate prenatal treatment if the outcome is positive. It is obvious that these measures, if generally carried out, would enormously reduce the incidence of congenital syphilis, and indeed they are reducing it.

The treatment of infantile congenital syphilis is attended with a smaller percentage success and a higher mortality than the late congenital syphilis. The organs of the newborn infant are permeated to a greater degree with spirochetes and there is less resisting power at this tender age. Nevertheless, modern treatment is accomplishing much more than was formerly achieved. For the newborn syphilitic infant I prefer intravenous treatment of the mother with neoarsphenamine or arsphenamine, the child receiving the effect through the maternal milk. I have seen an eruption disappear as rapidly after this procedure as if the infant itself had received the injection. At the same time the child should be given mercurial inunctions.

A little later, when the child has acquired greater resistance, the method of Fordyce and Rosen may be carried out with advantage, namely, the weekly intramuscular use of neoarsphenamine and, as an adjunct, the weekly intramuscular use of bichloride of mercury in oil. Two to three courses of eight injections each of the former are given during the first year, and courses of ten to twelve injections of mercury with rest periods of four to six weeks between the courses.[1] A special neutral neoarsphenamine is advised given into the buttocks in about 1 cc. of sterile distilled water. Sulpharsphenamine because

[1] Fordyce and Rosen advise the following dosage of neoarsphenamine:

First dose:

100 mg. for infants two to twelve weeks old.
150 mg. for infants three to nine months old.
200 mg. for children one to two years old.
250 to 300 mg. for children three years old.

Mercuric chloride dosage:

Gr. $\frac{1}{10}$, two weeks to six months old.
Gr. $\frac{1}{8}$, six months to one year old.
Gr. $\frac{1}{7}$, one to two years old.
Gr. $\frac{1}{4}$, over three years old.

of its greater tolerance by the muscle tissues has been advised as a substitute for neoarsphenamine. It has been used with reasonable success, but the advantage referred to is in considerable measure counterbalanced by a poorer influence on the Wassermann reaction. On experimental animals sulpharsphenamine is therapeutically inferior to neoarsphenamine. Where the veins are accessible, as they are particularly in late congenital syphilis, Stokes prefers neoarsphenamine or arsphenamine intravenously associated with inunctions or intramuscular injections of soluble mercury. I am decidedly in accord with the procedure of the intravenous route for the organic arsenicals. It is distinctly less painful than by intramuscular injection, and after a time most children offer little or no objection to it. All of the methods referred to are good and may be indorsed.

We have had a large experience, since its introduction, with bismuth, both in acquired and congenital lues, and I must confess to a decided preference for bismuth over mercury. This opinion is based both on laboratory study and on clinical observation.

Klauder, working in the Research Institute of Cutaneous Medicine,[1] found that the therapeutically active dose of potassium bismuth tartrate in aqueous solution or in oil was 50 mg. per kilo in rabbit syphilis. The maximum tolerated dose intramuscularly in rabbits is 125 mg. per kilo.

Nichols[2] determined that the maximum tolerated dose of bichloride of mercury intravenously was 1 mg. per kilo, and the therapeutically active dose 3 mg. per kilo. Schamberg, Raiziss and Kolmer[3] found the tolerated dose of bichloride of mercury by intravenous injection, in terms of pure mercury, to be 2 mg. per kilo.

Hopkins[4] demonstrated that potassium and sodium bismuth tartrate was tolerated intramuscularly in 75 mg. per kilo; the therapeutically active dose, *i. e.*, the dose capable of healing lesions and effecting a disappearance of spirochetes in rabbit syphilis was 2 mg. per kilo. He gives the therapeutic index as follows:

Bismuth	1 to 75
Neoarsphenamine	1 to 30
Arsphenamine	1 to 7
Hydrarg. salicylate	2½ to 1

[1] Arch. of Derm. and Syph., 1923, vii, 721.
[2] Jour. Exper. Med., 1911, xiv, 202.
[3] Jour. Cut. Dis., December, 1915.
[4] Jour. Am. Med. Assn., 1924, lxxxiii, 2089.

From this table it would appear that the therapeutic index for bismuth is even more favorable than for the arsphenamines. To be sure, the methods and criteria vary in different experiments.

Milian, of Paris, states that the spirocheticidal power of arsphenamine, bismuth and mercury is represented by the following figures:

Therapeutic activity:	
Arsphenamine	10
Bismuth	7
Mercury	4

Bismuth is believed by Milian to stand between the arsenicals and mercury.

Brown, Saleeby and Schamberg[1] compared the blood chemistry of rabbits treated with bismuth with the histological changes in the kidneys. They conclude that bismuth is a drug of relatively low toxicity. When one considers that 30 mg. per kilo, or nine times the maximum therapeutic dose for man, was injected into rabbits without producing an alteration in the blood chemistry indicative of nephritis, or histological evidence of material kidney damage, the latitude between the therapeutic and the toxic dose may be appreciated.

The laboratory results seem to be borne out clinically. Both in acquired and in congenital syphilis our results have been more successful since we have substituted bismuth for mercury. The conjoint use of neoarsphenamine weekly with weekly injections of bismuth has been highly satisfactory in acquired syphilis. The ordinary dose of the potassium tartrobismuthate for adults has been 100 mg., the same for children over eight; under eight, in general terms, 50 mg. Although thousands of injections of bismuth have been given, we have yet to see a serious complication.

I recall the instance of an eight-year-old child with an interstitial keratitis which was getting worse despite the fact that the little patient was at the time salivated with mercury. Immediate improvement set in after the inauguration of bismuth treatment, and later under neoarsphenamine and bismuth the keratitis cleared up, with restoration of vision. This combination in our clinic has been employed most satisfactorily in interstitial keratitis. Kleefeld in France believes bismuth to be superior even to the arsenobenzenes in keratitis.

Dr. Carroll S. Wright, in charge of the congenital syphilis depart-

[1] Jour. of Pharmacology, 1926, xxviii, 165.

ment of my clinic, has recently tabulated some 16 cases of late congenital syphilis which remained strongly Wassermann positive after ten to fifty intravenous injections of neoarsphenamine. Nine were reduced to negative with bismuth. In 1 case there was later a serologic relapse. Of 128 cases of adult syphilis, including latent cases, which had received one or several courses of neoarsphenamine, only 26 were Wassermann negative after an average of eighteen "neo" injections, some being treated also with mercury salicylate. Of the 102 positive cases, 52, or 51 per cent, became serologically negative after an average of fifteen injections of 100 mg. of potassium tartrobismuthate. We now employ these two medicaments conjointly. We are fond of using the syrup of the iodide of iron in congenital syphilis over long periods of time for its tonic and alterative effect.

Before concluding I wish to refer to another factor of importance in the treatment of syphilis, and particularly of congenital syphilis, namely, the maintenance and building up of the patient's resisting power. I agree with Stokes as to the importance of keeping syphilitic infants at the breast. Their chances of survival are distinctly better than when fed on cow's milk. Later on, care as to nutrition, hygiene, outdoor life and removal of focal infections must not be overlooked. Antibodies are formed in the system against the spirochetes, and the greater the native strength and resistance the greater aid will the physician secure through Nature's processes. Curiously our treatment of two chronic infectious diseases that have certain analogies is radically different. In tuberculosis we have sought to bring about arrest or cure by stimulating the defensive mechanism of the body. We are now searching for antituberculous medicaments as an aid in the warfare on this disease. In syphilis we have relied too exclusively upon specific drugs with insufficient attention to the patient's defensive reactions. In long-standing syphilis there is unquestionably a tendency on the part of the system to inhibit the action of Spirocheta pallida or to destroy it, and this is perhaps accomplished when vital organs are not too severely attacked. Warthin, as a result of extensive autopsy studies, believes that the spirochete may at times virtually be converted into a saprophyte. The treatment of congenital syphilis, therefore, should consist of a systematic and persistent attack on the invading parasite, keeping in mind, however, the aid to be derived from a maintenance of body resistance. These conceptions necessarily imply considerable individualization in therapeutic methods.

DISCUSSION ON THE PAPERS OF DRS. GREENBAUM, STOKES, MOORE, KOLMER AND SCHAMBERG

Dr. Francis X. Dercum: There are two or three points that have occurred to me as worth while mentioning. I am no longer active in hospital work, but in my clinic for many years we adopted as a routine method spinal drainage immediately after the intravenous salvarsan injections. In our experience, this method proved to be of decided advantage. We had a far greater degree of improvement in cases in which spinal drainage was practised. Spinal drainage unquestionably favors the introduction of the arsenic into the nervous tissues. The intradural pressure in both paresis and tabes is commonly very much above the normal. If this pressure be rapidly reduced, as in radical spinal drainage, the nerve tissue becomes correspondingly hyperemic and there is every reason to believe that this favors the penetration of the arsenic into nervous tissues.

Our experience has received confirmation in the experimental studies made by Dr. Hans Hoff (*Jahrbücher für Psychiatrie und Neurologie*, xlii, 1922–1923, p. 201). Hoff practised spinal drainage upon a large number of dogs, ten minutes after giving intravenous salvarsan injections. He found that he could recover arsenic from the nervous tissues invariably under these circumstances. He could recover none from the nervous tissues in experiments in which spinal drainage was not practised. He could recover arsenic freely from the other viscera. In our clinic we observed the most remarkable improvement, *i. e.*, a cure (?) or at least remission of great duration. In one instance of paresis in which the symptoms both mental and physical had become well established, the man underwent a degree of improvement which enabled him to return to the control of a large industrial concern and in this, after four years, he is still fully active and most efficient. We should be very careful, of course, in judging from our results because, years ago, before the introduction of salvarsan, long periods of remission were often established by simple physiological methods of treatment, *i. e.*, by rest in bed, full feeding, massage and tonics. However, the results with salvarsan injections, accompanied by spinal drainage, have in our hands proven to be of such value that I believe that spinal drainage should be generally practised. It can do no harm; it can only do good.

In the way of supplementing Dr. Schamberg's remarks, I would call attention to the fact of the frequency with which positive Wassermann tests are met with in dementia præcox. Indeed, I believe that an accurate statistical study would reveal approximately 33 per cent.

Dr. Alfred Gordon: I will confine my remarks exclusively to neurosyphilis. I was somewhat surprised in relation to the talk of treatment that no word was mentioned of the treatment of neurosyphilis with intramuscular injection of neoarsphenamin. For a number of years —at least ten or twelve—I practised all methods of treatment of neurosyphilis, namely, Swift-Ellis, intravenous and finally intramuscular injections. In comparing the results I found that with the intramuscular injections

the results are just as good as with the other methods. The great advantage of treating with intramuscular injections is that you commence with extremely small doses and repeat frequently; I commence with 15 cg., giving every other day. The tolerance is remarkable. The patient is able to go about his business, by coming for a few minutes to get the injection, but the injections have to be extended over a number of months. This method has also the advantage of testing the individual idiosyncrasy. Another thing that struck me tonight was the absolute faith in the Wassermann reaction. My personal experience in the treatment of neurosyphilis is that I found in many cases while serological findings were still positive I nevertheless got good clinical results. There was a striking want of parallelism between the two. A positive Wassermann is corroborative, of course, but many cases of headache, of generalized epilepsy and even epilepsy of the Jacksonian type of hemiparesis, etc., have improved by the treatment I have outlined without having any influence on the Wassermann reaction. In regard to what Dr. Kolmer said of marriage, he spoke almost exclusively of syphilis in husband or wife as a recent infection. What about cases of very old standing where the wife or husband had the infection twenty or twenty-five years before marriage? I have at the present time about 36 cases of conjugal syphilis. For example, a man who began to show symptoms of tabes long after he married, and then years later, eight or ten, the wife began to show symptoms of neurosyphilis. This incidence opens the question of the existence of neurotropic and dermotropic treponema. The subject is extremely important as it has a social bearing. I would ask Dr. Moore if he had observed any trouble with the eyes in using tryparsenate, because cases of real blindness and permanent damage have been reported?

Dr. Solomon Solis-Cohen: I wish to thank Dr. Stokes for his very clear exposition of the subject of cardiac and pulmonary syphilis. I believe this will do a great deal of good, especially in Philadelphia, by recalling attention to the subject and emphasizing the frequency of such cases. Cardiovascular syphilis is perhaps nowadays freely recognized, but many physicians seem reluctant to accept the diagnosis of pulmonary syphilis; while those who were taught to make such diagnoses find increasing occasion to do so. There are many cases in which the *x*-ray reveals changes in the lung pictures which are interpreted as postinfluenzal and sometimes as tuberculous, which are in reality of syphilitic origin.

I did not intend, however, to discuss the diagnosis or to attempt to add anything to what Dr. Stokes had said, merely to reëmphasize the importance of the subject. I would like to ask of all the speakers what their opinion is of a treatment very much in the journals at the present time, but which has not been alluded to in the papers, namely, the use of indifferent proteins, especially of milk, to produce a febrile reaction.

Dr. Moore (closing): In regard to the remission of syphilis, the literature has been enriched by two recent exhaustive papers, one in this country and one in Germany, and a summary of the experience of a great many people seems to be that in untreated paresis they expect 10 to 15 per cent of

spontaneous remissions. These remissions last from two months to sometimes longer than a year. However, in the method of treatment which I briefly touched upon, it is possible to obtain remissions in early paresis as high as 50 to 60 per cent of cases, and in all cases in which we used malarial organisms as high as 30 per cent.

Dr. Gordon brought up the question of repeated doses of arsphenamin. I said nothing about this treatment because I have had no personal experience with it. I endeavored to confine my remarks to the treatment personally used. The results I presented to you were not excellent serologically. The term excellent is used to indicate clinical and serological results. It is evident that patients may present a good serologic reaction and a poor clinical picture; such a result cannot be classed as excellent by any stretch of the imagination.

As to tryparsenate, in common with others who have used the drug I have seen damage to the visual apparatus. There have been 2 or 3 cases reported. In general, in patients who are not mentally so clouded as to prohibit any coöperation the possibility of damage is not great. It is possible to determine the onset of visual difficulty in early stages and stop the drug before harm.

In regard to Dr. Cohen's question, I have had no experience with nonspecific protein therapy, except in so far as the central nervous system may be alive to nonspecific protein treatment. One German observer has reported very interesting results in paresis following the muscular injection of milk. So far as I know, his clinical results have not been elsewhere confirmed.

Dr. Schamberg (closing): Referring to Dr. Cohen's query concerning the influence of foreign proteins on syphilis, Dr. Greenbaum has been interested in this work and I have seen cases of gummatous ulceration in my clinic disappear under the injection of milk protein. In persistent positive Wassermanns we have used injections of milk protein, in some instances with good results, in some other instances without results.

I would like to refer to another question, namely, the use of the term hereditary syphilis, or heredosyphilis. I believe that these terms should be abandoned. From the biologic standpoint the term heredosyphilis or hereditary syphilis is a misnomer. There is really not much more justification for speaking of heredosyphilis than there is of speaking of heredosmallpox, because we know children are born at times with the smallpox eruption and at times with scars resulting from an intrauterine attack. From the biologic standpoint no infectious disease is hereditary.

THE DIAGNOSIS OF TRACHEOBRONCHIAL TUBERCULOSIS*

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The literature of tracheobronchial and "hilum" tuberculosis is voluminous. The major part of it deals with physical signs and symptoms assumed to be associated with this condition. The descriptions of the actual lesion as demonstrated by roentgen ray are somewhat vague both as to site and appearance. Not a few observers report calcification due to tuberculosis in the majority of all patients, whether or not they have been exposed to an open lesion. Many are governed in their recognition of the pathologic and anatomic lesion by the presence of a tuberculin reaction, and the association of evidence such as underweight, fever, cough, night-sweats, fatigue and dyspnea, which are generally assumed to be allied with and to result from the lesion. It is desirable to consider the possibility of consistent and reproducible demonstration of tracheobronchial tuberculosis. If we are to understand its pathogenesis and symptomatology, those cases showing roentgenographically reproducible lesions must be studied apart from the group whose anatomic diagnosis has rested on various symptoms. As has been shown elsewhere[1] these lesions are frequently the forerunners of diffuse pulmonary infiltrations. While there is no evidence that it is the source of these infiltrations, and much that it is not,[2] tuberculous tracheobronchial disease is an indication of severe infection, and such infection may not only be carried to the lymph nodes but may also remain latent in the lung and develop after the glandular disease gives evidence of healing.

The matter of roentgenographing the lungs and of determining what abnormal changes could be conclusively recognized has

* Read November 3, 1926.

occupied the writer for some seven years. The subject was approached in two main studies, one to discover what elements of the lung structure might be differentiated roentgenographically under the simplest conditions, the other to provide against the effect of cardiovascular movement in disturbing lung detail. The mechanism, methods and some conclusions of the latter study[3] have already been briefly reported.

The former aim was sought by roentgenographing the excised lung, and then comparing the specimen, area by area, with the roentgenogram. Some 400 pairs of lungs were thus examined, about 150 of these being maintained inflated during exposure. Excised lungs have the advantage of relative availability and ease of handling; and slightly finer detail may be shown when the ray can be decreased to what is just sufficient to penetrate the lung itself. Furthermore, no contiguous structures, such as spine, mediastinum and sternum, complicate the shadow, a matter of importance in discovering the roentgenographic demonstrability of normal and pathologic lymph nodes of the tracheobronchial and intrapulmonary groups. In this paper the lymph nodes lying within the lung will be described as intrapulmonary, those outside as tracheobronchial. The division is artificial pathologically but convenient for anatomic and roentgenologic descriptions.

In passing it may be noted that in this series of pathologic specimens, which was examined by Dr. Opie, the pulmonary primary lesion was demonstrated in the lung substance in all but about 1 per cent of the cases in which a glandular lesion was observed. It was at least as frequent that a pulmonary primary nodule occurred without a glandular lesion as that the reverse was found. Below are given the anatomic and roentgenographic characteristics by which particularly the lymphatic element of the associated primary pulmonary and glandular lesions[4] may be conclusively recognized. But the criteria apply equally to those pulmonary primary lesions which are situated in the parenchyma close to the main stems, for example on the medial aspect of the lung, subpleurally at the inner part of the interlobar fissures, or, more rarely, in the lung substance which lies about the main stems. Frequently the pulmonary primary lesion is larger than the glandular (Fig. 9), and constitutes a greater menace; frequently it progresses into a diffuse or localized infiltration, usually nonapical because the originating primary foci, distributed in proportion to lung volume, occur more commonly

in the lower part of the pulmonary cone. But from the diagnostic point of view, the pulmonary primary offers few difficulties in recognition because it is usually situated subpleurally[4] and therefore occurs chiefly at the periphery (Fig. 9). It is, in consequence, rarely to be confused with or obscured by trunk shadows, and is obvious, unless it lies behind the cardiac or diaphragmatic shadow. Here too it may be demonstrated, if calcium-bearing, by the proper quantity of roentgen ray. And even when calcium-free, if it lies clear of other densities, the pulmonary lesion can be recorded at least as well as the vascular branches of supply in the area in which it is situated. Excised lung studies show the calcium-free pulmonary primary to be as dense, per unit of volume, as artery or vein and consequently, when synchronization permits the recording of fine vascular detail, any equally fine pathologic detail will be shown at least as well. Subpleural densities are remote from the effect of cardiovascular vibration, described later, and if anterior, are especially favorably situated for accurate representation.

The term tracheobronchial tuberculosis is preferable to hilum tuberculosis because, as pointed out by Opie, the former describes accurately the glandular element of the complex of primary infection. The lesion rarely breaks through the gland capsule, and if it does the rupture is usually into a bronchus, artery or vein. Direct progression through the capsule into the areolar tissue of the hilum was seen only once in a series comprising over 400 specimens, including many fatal infantile cases. While infiltrations occur involving the parenchyma near the main bronchial and vascular stems, in our studies, both of postmortem material and of living persons, it could be shown by careful examination that there was also an apical lesion, and the presumption is that, for the most part, localization near the root is merely one of the less common manifestations of bronchogenic extension. Furthermore, it was notable in the excised lung series that frequently, in the presence of a slight apical lesion, a more definite infiltration in the upper part of the lower lobe or the anterior lappet of the upper lobe gave the roentgenographic appearance sometimes described as hilum tuberculosis. Even in good stereoscopic films in the living it is not always possible to define the plane in which a lesion lies. Section will decide the question in the excised, and variations in the patient's position will solve it in the living.

The postmortem material for this study comprised many lungs

free from gross lesions or even agonal edema. From these a good conception was obtained of the number of trunks that might be recorded. They are strikingly more prominent (Fig. 1[5]) and numerous than in the films of normal persons in life, and more bronchial bifurcations, the site of nodes are rendered visible. On section of the lungs special attention was directed to the possibilities of roentgenographic localization of the lymph nodes, both the normal and those showing various stages of fibrous and inflammatory change. Meticulous study of the films was made in cases showing diverse types of nodes, from the dense, fibrous, coal-pigmented lymph node of the city dweller, and the succulent nodes of acute, nontuberculous bronchopneumonia, through the stages of fresh semifluid and old puttylike caseation, to the stony nodules and masses of dense calcification. In summary, it was found, as regards the nodes of the trachea and extrapulmonary bronchi, that only when calcium infiltration of a necrosed area was present was there any distinctive shadow. As regards lymph nodes within the lungs, it was found that the presence of calcium was the only roentgenologic indication of the presence and site of a lymph node, except for that segment of the noncalcified margin of such large calcium-bearing glands as projected laterally to the main stems and larger branchings of the air and blood-bearing trees and so contrasted with pulmonary parenchyma. Intrapulmonary nontuberculous glandular enlargement sufficient so to project was not seen, although there were many specimens showing large, succulent glands due to acute infections. Such glands did not throw shadows to be differentiated in the composite shadows of the main stems and their branches which cross and surround the glands, and this study affords us no reason to suppose that they may be recorded in the living, or confused with tuberculous disease. While this finding is contrary to the inferences to be drawn from many articles, it is in harmony with the properties of the roentgen ray, which is obstructed, per unit of volume, only in relation to atomic weights. Furthermore, although many roentgenograms were made during and immediately after acute, often serious, infections in children at all ages, in no case was there evidence of roentgenographic demonstrability of glands of the tracheobronchial group referable to the acute infection, that is, calcium-free.

Reference to Figs. 1 and 2 (see also Fig. 1[5]) will show how well adapted to the fullest representation of any normal or abnormal

density was the material studied and the method of making the stereoroentgenograms. The trachea and bronchi with their sheathing tissues stand out clearly. Except for the surrounding areolar tissue and occasional tags of the great vessels, all that might obscure the lymph nodes was removed. The lungs were inflated until the air entered everywhere, then allowed slightly to subside so that the pleura should not be unnaturally tense. The total volume of air contained, and consequently the basis for securing contrast, was similar to that existing *intra vitam*. The lungs were allowed to hang freely, and it was sought to keep them in a position as nearly normal as possible merely by their apposition to the front of the cassette changer. Occasionally string or gauze was used to separate the bases. Stereoscopic exposures were made, the duration varying from one-fiftieth second to one-twentieth second; rarely as much as one-tenth second was used. The photographic effect sought was that giving the greatest detail. The relation of the quantity of exposure to that of the living was about 60 to 75 per cent. The absence of the chest wall reduced the amount necessary. It was found that full exposure, as for the living, marred the detail in the periphery but that otherwise the films were similar.

Clearly, tracheobronchial lymph nodes, both those lying immediately outside the lung and those further cephalad on the bronchi and trachea, are favorably situated, in the excised lung, for the fullest demonstration of changes in contour and density. Yet in all the specimens studied partial outlines of only two lymph nodes could be made out lying within the shadow cast by the ensheathing areolar tissue. In all other cases, apart from calcium infiltration, when the site of nodes could be determined from the film, it was due to enlargement; whether the cause of the enlargement was coal-pigmented fibrous tissue or acute edematous swelling, its indication of the site was due to the size, not to the intrinsic density of the node. That is, only in instances where the node projected laterally from the trachea or bronchi, bulging out the sheath of areolar tissue, was its contour indicated. Otherwise, even if a node contained calcifications or caseation impregnated with calcium, the shadow recorded conformed only to the outlines of the calcium-bearing lesion. The peripheral lymph tissue, whether hypertrophic or fibrous, and the gland capsule were not dense enough to cast a shadow differentiating them from the contiguous areolar tissue. Glands containing soft fresh caseation due to acute, rapidly advanc-

FIG. 1.—Right lung: The vessels were not tied before section. Inflation expels most of the unclotted blood and these lungs contain little. Neither the arterial nor the venous main stem can be seen. On section, the upper and middle lobes are dry, the lower red and contains fairly abundant fluid. At the hilum, the carina and to the right of the trachea are hard, anthracotic, fibrous lymph nodes containing hard calcified masses. Left lung: The left lung resembles the right. There is a firmly calcified nodule in the upper lobe. The nodes at the hilum resemble those on the right. There is no evidence of the presence of the uncalcified part of nodes except for the calcium-containing bulge above the left bronchus. The fibrous, pigmented glandular margin is no denser than the ensheathing areolar tissue.

FIG. 2.—The vessels were not tied before removal of the lungs. There is a dense calcium shadow at the carina, two others lateral to the right main stem bronchus and one in the substance of the right lung. The glands containing the calcium at the carina and on the bronchus are not distinguishable, although the one at the carina is particularly favorably situated. The fibrous capsule enclosing the mass in the lung substance throws a faint halolike shadow.

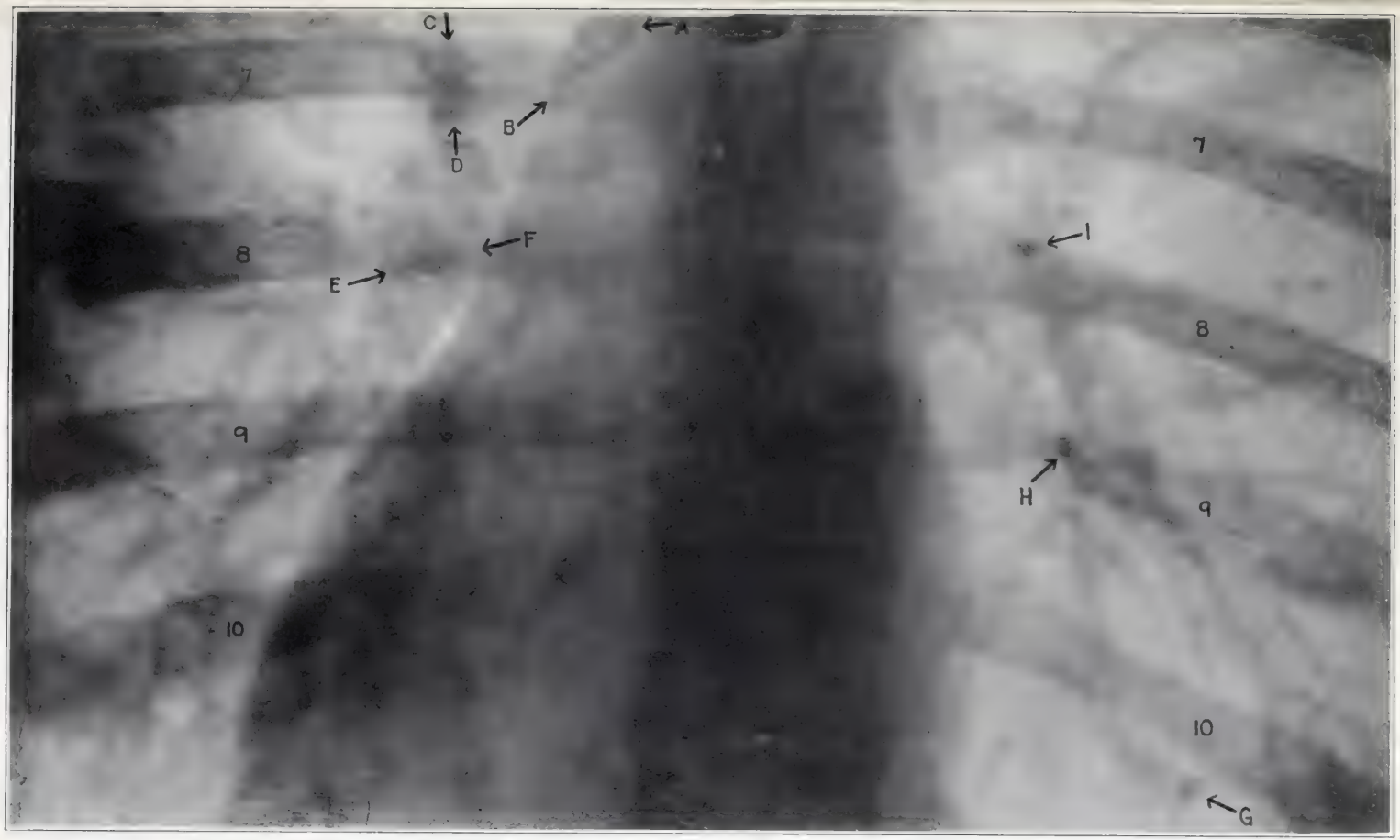

FIG. 4.—A postero-anterior view. In the right tenth interspace, *G*, is a rounded density, due to a vessel directed toward the target, that is, axial to the incident ray. Similar densities, *H* and *I*, appear on the arterial main stem shadow. On the left seventh rib is a dense, irregular, club-shaped shadow, *A B*, 1.7 x 0.7 cm., head touching the aortic contour. One centimeter lateral to its lower end is another dense shadow, *C D*, 1.3 x 0.6 cm. In front of the eighth rib, on the arterial main stem shadow, is a granular density, *E F*, 1.7 x 0.8 cm.

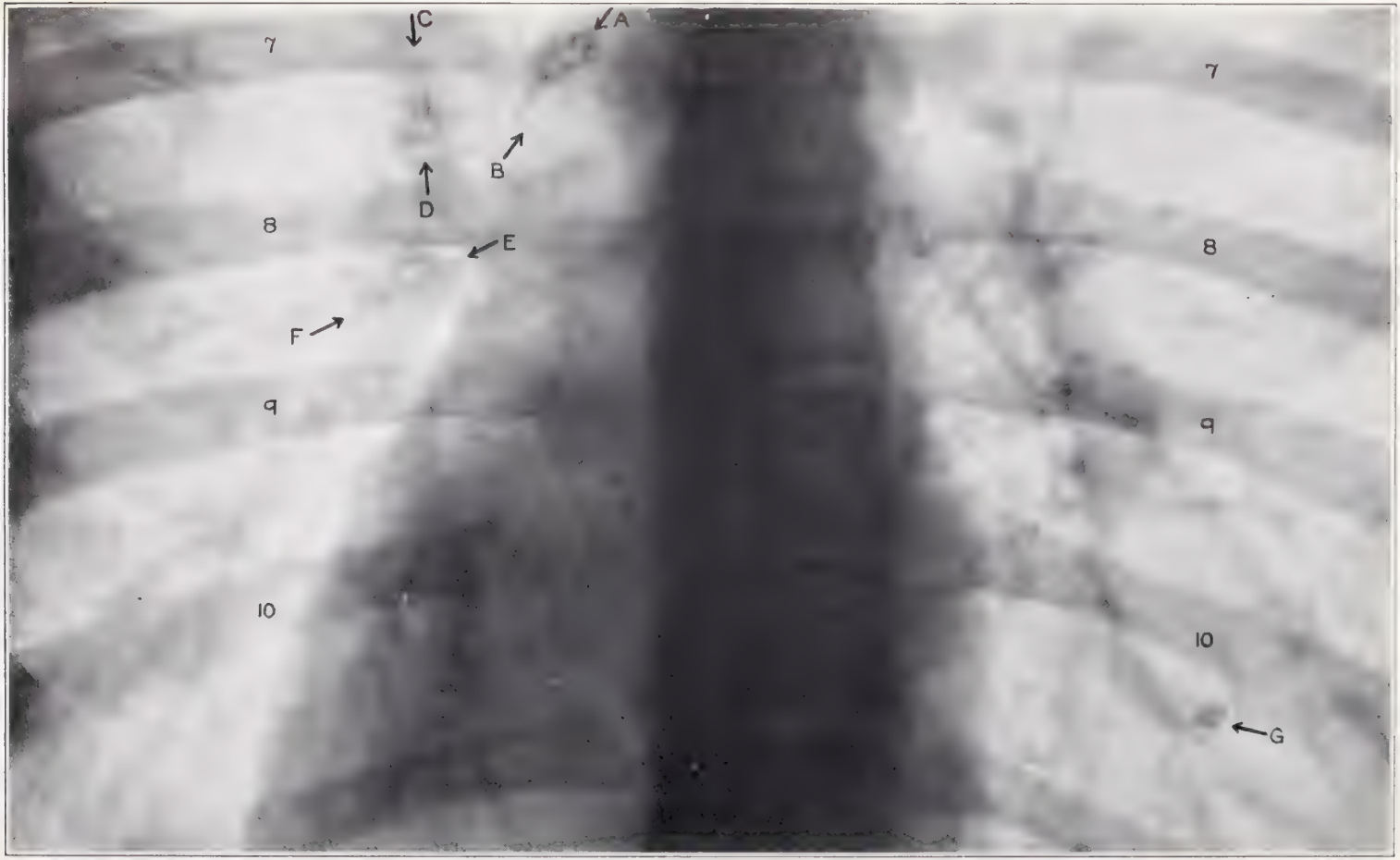

FIG. 5. *A B, C D, E F,* and *G* are still seen in this exposure made at the lower stereoscopic level. *H* and *I* are not clearly recognizable. *E F* is better seen below the eighth rib shadow, than in the former figure, in which it lies in front of the rib.

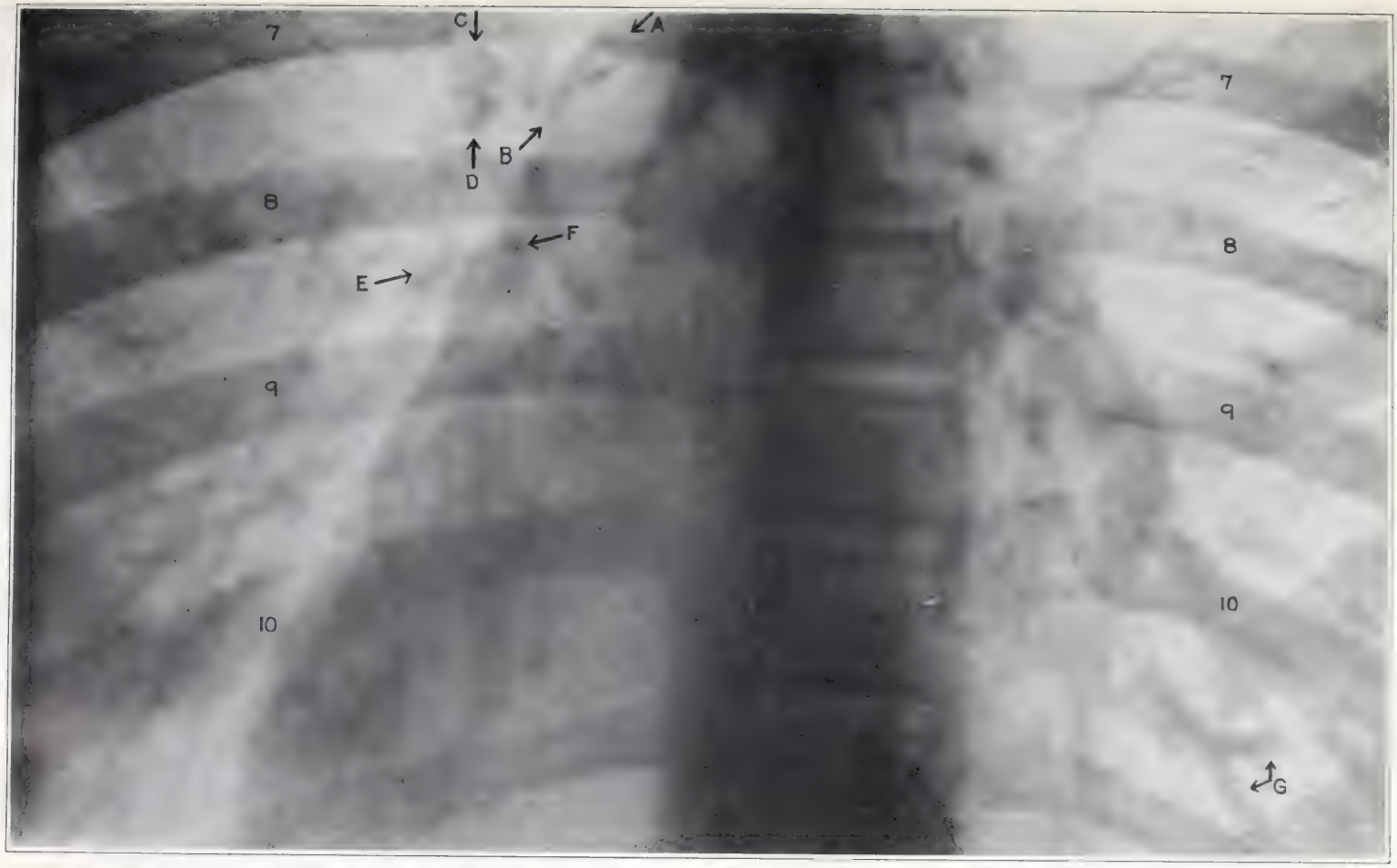

FIG. 6. A rotation exposure. The rounded spot *G* in the right tenth interspace is replaced by a Y-shaped vesel, the upper arm of which, axially radiated, caused the density *G*. The lesions on the left can be recognized as persisting. *C D* is more clearly defined, seen from the new angle. *E F* in the eighth interspace is now partly behind or upon the cardiac shadow.

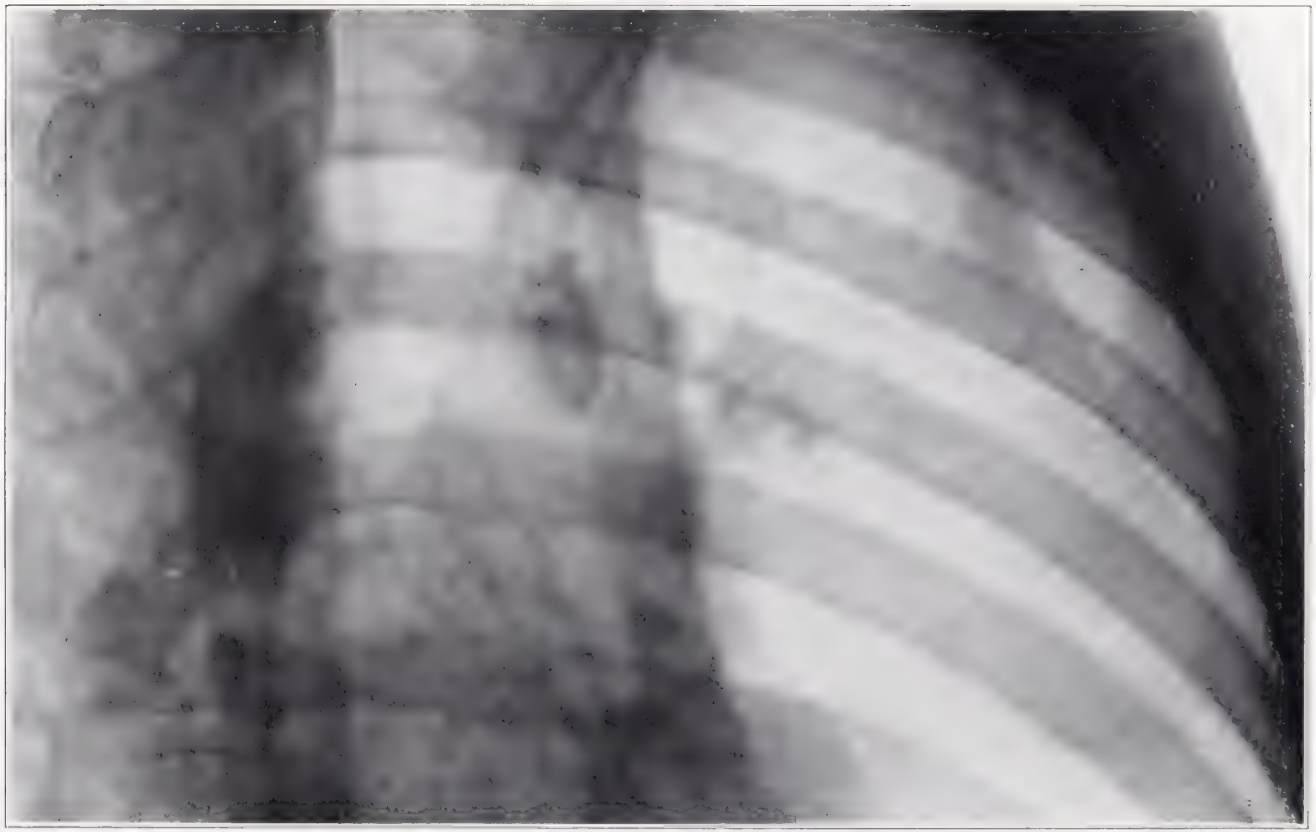

FIG. 7.—This patient's father had positive sputum in February, 1924, and was at home almost continuously until his death, in February, 1926. In this oblique view a granular shadow, 2.2 x 1 cm., can be seen just to the right of the carina. This is due to a relatively light calcium infiltration in a large necrosis. There was no mediastinal bulge.

FIG. 8.—Left anterior oblique exposure of a boy, aged seven and a half years, in contact with a tuberculous father (sputum positive) during four and a half years until one and a half years prior to this roentgenogram. Close to the right side of the trachea, above the carina, at the level of the fifth rib and interspace and the sixth rib are two oval outlines. The lower pole of the upper one, the long axis of which is indicated by *A*, overlaps the upper pole of the lower one, the long axis of which is indicated by *C*. Their lateral borders cross at *B*. The lower one is not unlike the nucleus of an epithelial cell, with a dark, oval outline and an irregular dark spot within it, at the fifth interspace level. *D*, *E*, *F*, mark the upper pole, posteromedial border, and lower pole of another oval density lying posteromedial to the right bronchus below the carina, which is crossed by the sixth rib. These densities are due to scant calcium infiltration of caseous glandular necrosis. Neither of these shadows could be seen in the postero-anterior position. The outlines of the glands enclosing the calcium-bearing necrosis can be seen neither in the illustration nor in the film from which it was taken. There was no mediastinal bulge. The left bronchus passes in front of the spine at the level of the seventh rib. The lower end of the right bronchial main stem is marked by *G*. The patient was well nourished and active, pulse and temperature were normal. There were no complaints nor symptoms

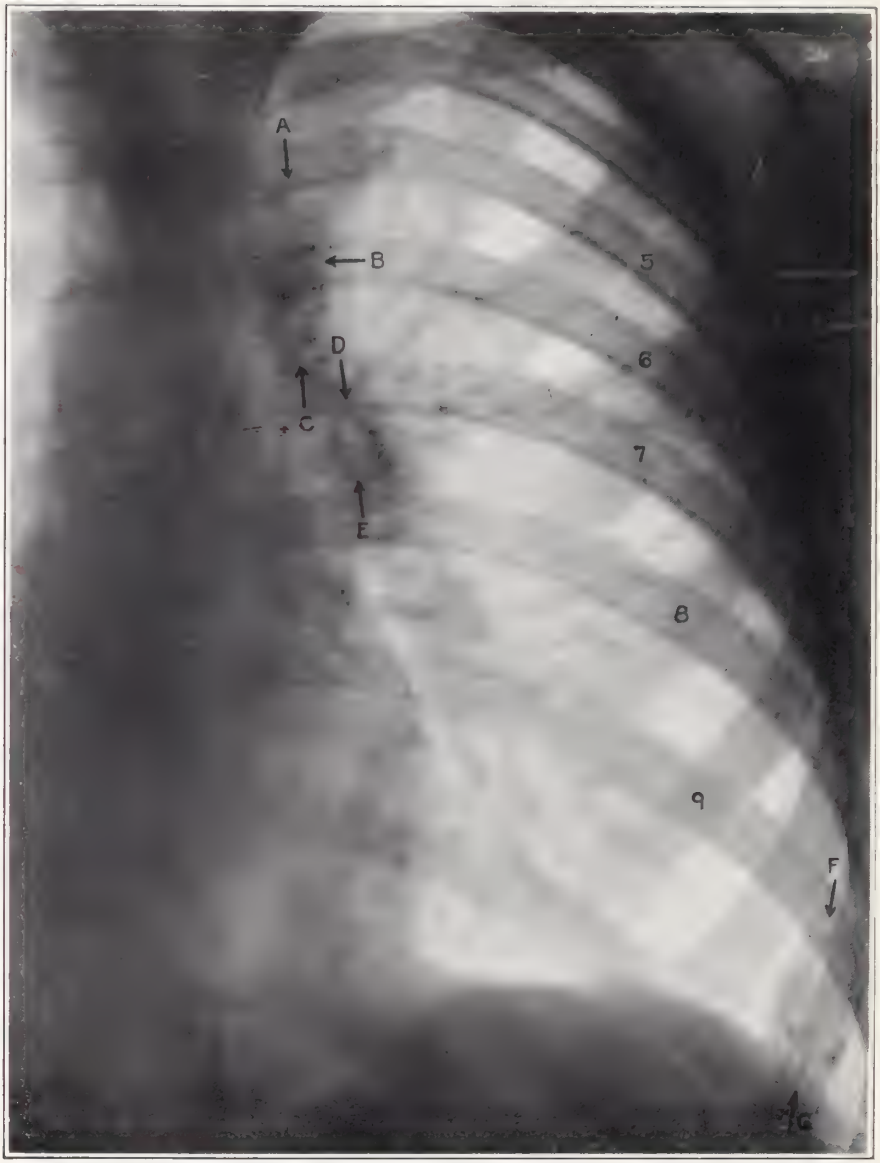

FIG. 9.—Patient, aged five years. Exposed to open lesion of mother for about three years. Child is apparently well. The patient is almost in an oblique position in order to throw the mediastinal lesion clear of the spine. The head of the left clavicle is behind the spine; that of the right lies lateral to the spinal right border. In front of the ninth rib and interspace and the tenth rib is a pulmonary lesion *F G*, 2.6 x 1.3 cm., lightly infiltrated with calcium. On the arterial shadow in the seventh interspace is a calcium shadow, *D E*, 1 x 0.8 cm. Lying on the right tracheal wall in front of the fifth interspace, sixth rib and interspace and seventh rib is a similar shadow *A B C*, 2.3 x 1.3 cm. The trunk markings reproduce faintly, but a uniform and regular radial striation may be seen. There is no shadow of diffuse pulmonary infiltration.

ing infections were similar in density to nonspecific edematous nodes. They were distinguishable only if large enough to bulge out the silhouette of the areolar mantle (Fig. 1).

In discussing the recognition of intrapulmonary lesions, it is necessary to consider at each point what modifications of the appearances seen in roentgenograms of the excised lung are effected by cardiovascular movement in films of the living. The intrapulmonary nodes and lymphoid masses, situated at the forks of branchings of the bronchial tree, are surrounded by structures of roentgenographically equal density. In proportion as one approaches the hilum and the nodes are larger, they are crossed, surrounded and overlaid by arteries, veins and other bronchi converging on the main stems. The vascular trunks, at least, are, per unit of volume, as dense to the roentgen ray as are normal nodes, or, indeed, any nodes, without calcium infiltration, as pointed out above. The glands most frequently and severely infected, those which lie just lateral to the main-stem bronchus (Figs. 1, 2 and 4), that is, on the side whence the bronchial trunks arise, are not only crossed by the venous trunks but have superimposed on them the main arterial stem. In order for nodes so situated to be recognizable roentgenologically they must, unless containing calcium, project not only beyond the shadow of the main arterial stem but also beyond the zone where the crossing of the converging vascular trunks forms a complex, unequal shadow, for only so can they be shown in contrast to the air-bearing parenchyma. In the living this zone is particularly confusing when the exposure is as long as the cardiac cycle, because of the effects of cardiovascular movement; or, when a shorter time is used, if the exposure chances to occur during systolic movement. Diastolic rapid exposures (not more than 0.05 second at pulse-rates about 100), are best adapted to show the origin, course and branchings of trunks, and to differentiate abnormal glandular enlargements which may project into this zone.

We have seen, in our excised lung series, no instance of a noncalcium-bearing gland projecting beyond the arterial main stem shadow, and only one instance in the living. In this case repeated, but not yet protracted, observations, suggest a tuberculous etiology. Not infrequently a calcium-bearing gland is seen to project, and to show, laterally, a margin surrounding the calcium-bearing necrosis, its peripheral contour standing out against the less dense pulmonary parenchyma into which it bulges. It is somewhat more common to

find such a gland to lie wholly medial to the lateral margin of the arterial main stem. Then only the calcified area is visible (Figs. 4, 5 and 6), the remainder of the gland being no denser than the arterial main-stem shadow and the maze of vascular trunks abutting on it and crossing it in many directions.

A common source of diagnostic error is the sharp, round shadow cast by a vascular trunk directed toward the target so that the incident or primary ray is approximately axial (Figs. 3, 4, 5 and 6); or the semioval shadow often recorded when the plane of the curve

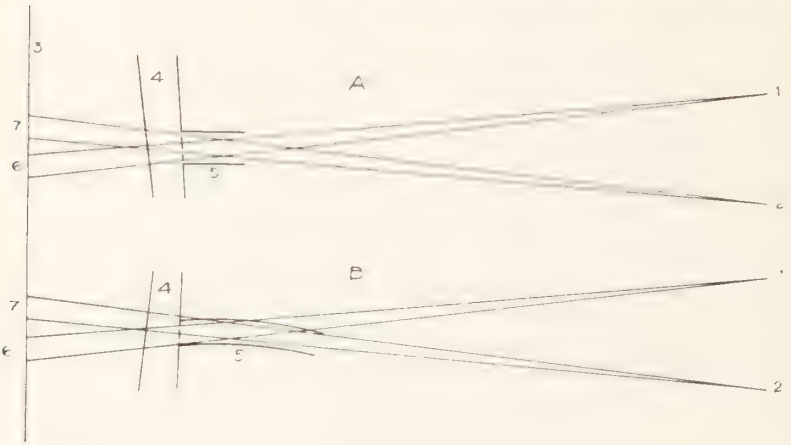

FIG. 3.—The stereoscopic drop is exaggerated to facilitate drawing. Actually the two rays intersect at a more acute angle and may consequently be practically axial to narrower vessels. 1, Ray from upper position of tube; 2, ray from lower position of tube; 3, stereoscopic films, represented as one; 4, vessel directed in plane perpendicular to axial rays; 5, axial branch; 6, shadow cast on film 1; 7, shadow cast on film 2.

of a vascular trunk is directed slightly above or below the axis of the ray. Each of these is comparable to and often situated beside the dense ring-shadow due to a bronchus directed toward the target. The former is more common and may occur anywhere in the lung field except at the apex and the extreme lateral margin, inasmuch as branching of trunks continues well out to the periphery. In proportion as caliber diminishes these shadows of vascular causation are smaller peripherally. Those trunk shadows radiating from the hilum in planes nearly perpendicular to the incident ray often show the branches which arise from them axial to the ray as more or less well-defined dots particularly in the middle and outer

third of the lung field (Fig 1[5]). Many of these dots are ill-defined and blurred when exposures occur during systolic movement and have been interpreted as thickening of the trunk on which they appear. The apical branches of supply, however, running straight into a rapidly narrowing cone, are little disturbed by vascular movement and frequently show these dotted vessels.[6] Thus branches arising parallel to the axial ray are responsible for many "beaded" trunks and practically always for the so-called "capped" trunks, which have been interpreted as due to calcium in a lymphoid mass at the bronchial bifurcation. In films of the excised lung, when the vessels are tied off before removal, the number of these round and oval shadows along the course of the vascular trees is amazing, and their nature may be verified by section. Practically, both for the excised and the living, the diagnosis may be made conclusive by appropriate methods. The proof lies in roentgenographing the beaded trunk from a slightly different angle, when it is seen that the dots have disappeared and that branches arise from their site. On the other hand, a calcified lymph node or pulmonary nodule may show a variation in contour but will not be lost by this procedure (Figs. 4, 5 and 6). For many such determinations the distance of the stereoscopic shift supplies the necessary new angle of radiation, when the films are truly stereoscopic, that is, when the lungs are undisturbed by changes in relative cardiac volumes and by differences in pressure in the elastic vascular tree.[6] In other instances the conclusive differentiation of vascular artefacts from calcium-bearing lesions by stereoscopic exposure alone is not possible. It is apparent that even with a full stereoscopic drop and rapid diastolic exposures, the incident ray from both the target levels will be practically axial to the shorter, thicker trunks that run parallel to it (Fig. 3), or even to those narrower trunks whose natural curve forms an arc to the axial rays, so that at both stereoscopic levels a dense column of blood is opposed to the ray. The former condition frequently obtains upon and close to the arterial main stem, and medial to the main stem bronchus, due, respectively, to arterial and venous axially directed branches. Such vascular artefacts are especially disturbing in films of infants, and troublesome in those of older children. In infants the trunks closest to the main stem cause the artefacts, and these are large enough in proportion to the chest to suggest serious lesions. The older children show more numerous artefacts, since there are more vessels large enough

to throw a shadow. Often in children of ten to fifteen years the very number and the uniformity of size, quality and distribution of these shadows is enough to cast doubt on their having a pathologic significance.

The type of vascular artefacts due to a curving vessel axial to the incident ray is seen commonly at the posterior level of the fifth and sixth interspace, right and left.

It was found that exposures made at various points above and below the usual stereoscopic upper and lower levels commonly failed to solve the nature of these questionable shadows. This is due to the large number of branches arising at every level from the main vascular stems. Thus one could not be sure, even after a shift, of, say, 7 inches below the usual stereoscopic midpoint, if a persisting density were not simply a new vessel coming off lower down. It was then found that a slight rotation of the patient about his perpendicular axis was of great value in deciding the question. After many trials, I have sought to rotate the patient so that the right cardiac border coincides in the film with the right border of the spine. A single exposure so made at the lower stereoscopic level has enabled us to determine the identity of practically all densities lateral to the spinal border, right and left (Figs. 4, 5 and 6). Only very occasionally is it desirable, chiefly for the diagnosis of densities lying near the bronchus to the left upper lobe, to rotate the patient in the opposite direction (that is, the right shoulder and hip away from the cassette changer, and to the same degree as for the above noted left-shoulder-off position).

The addition to the usual stereoscopic technic of the first described left rotation (left side away from the film) is of value not infrequently in the diagnosis of lesions sufficiently unstable to warrant careful oversight and reduced exertion to ensure their healing. Many intrapulmonary unencapsulated caseous necroses, containing only delicate and diffuse calcium infiltrations, can be certainly diagnosed by the addition of slightly rotated exposures. A true lesion will continue to be recorded in the rotation film as a shadow of constant quality, finely and irregularly granular, or made up of softly lamellated and crenated lines (Figs. 4, 5 and 6), or irregularly stippled; and its contour will show such slight changes as will be harmonious with that in the postero-anterior position. Artefacts, on the other hand, are resolved into their components or entirely disappear. Especially valuable is the rotated position in differ-

entiating from true lesions the very common densities in the right cardiophrenic angle due to venous trunks, and the numerous spots in the inner third of the lung field due to arterial and venous branches. It is also often useful when it is of moment to diagnose densities in the outer half of the lung field. Naturally, as the caliber of the vessels diminishes peripherally, lesions likely to be confused with densities of vascular origin are smaller and of less clinical importance. Care must be taken not to turn this exposure into an oblique (see below), for in the oblique position a new and, for this purpose, confusing number of rounded densities occur, and it is difficult to determine which shadows one was seeking to resolve.

In many cases a position midway between the postero-anterior and the lateral is indispensable. In the roentgenographic recognition of tracheobronchial tuberculosis in the living, the shadows of spine and sternum offer a serious difficulty. Without the oblique exposure it is impossible to recognize clearly (see Figs. 7, 8 and 9 or even to suspect the presence of fresh and unstable tuberculous necrosis. Many calcium-bearing lesions, both at the carina and to the right of the trachea, measuring up to 2.5 cm. across, completely escaped demonstration in the postero-anterior position (see Fig. 8, *D*, *E*, *F*). It is perhaps important here to emphasize what has been said above with reference to the excised lung, that calcium-free caseation, apart from mass and consequent projection into the lung field, is roentgenologically indistinguishable from the areolar tissue and vessels of the mediastinum. Caseation so extensive as to bulge into the lung field, a condition chiefly seen in infancy, must almost invariably prove fatal. In the living it is rare that a calcium-bearing tracheobronchial gland can be seen to project beyond the margin of the spinal shadow. We have not seen tuberculosis of the tracheobronchial glands cause a uniform, rounded mediastinal mass except in fatal infantile cases. A postero-anterior film showing smooth and oval mediastinal bulges should be scrutinized for distortion of the patient's position by which the shadow of the sternum appears to the right or left of the spinal shadow. Malignant tumors will rarely come in question in children. Rotation is probably responsible for the majority of apparent mediastinal enlargements. It is in infants and in children up to four years that most "broadened mediastinums" have been interpreted as pathological. Apart from errors due to rotation and to thymus shadow, exposures made during systole and during expira-

tion are frequent causes of broadening of the mediastinum. After the fourth year response to the request for holding the breath is obtainable, and broadened mediastinum is rarer.

The chief value of the oblique position is that one is able to record the trachea, the carina and the two bronchi as far as their entrance into the lung in a way that is not possible when they are situated between the spine and sternum. Calcium-bearing necrosis throws its characteristic shadow, and while the gland in which it lies is not to be outlined, from the nature of the granulation, whether faint and small, or large, dense and confluent, one can estimate the condition and lability of the lesion. The longer calcium infiltration has endured, the better are the chances that the lesion has been walled off. We have, in three years' observation, of cases reported elsewhere[1] found no evidence of reabsorption of calcium from glandular lesions. In several cases the shadow has become heavier and better delineated. In so far as the glands are concerned, caseation, if accompanied by calcium infiltration, appears to remain. In view of clinical and experimental evidence[2] that reabsorption of caseous pulmonary and glandular infiltration occurs further and more extended observations on calcified glandular caseation are desirable.

The situation, extent and contour of the calcium-bearing glandular lesions have been of interest. As pointed out by Opie,[3] the common lesion in a lymph node above the carina (Figs. 1, 7 and 8) is almost always situated to the right of the trachea irrespective of the side in which the pulmonary and intrapulmonary glandular lesions occur. In the oblique view, it may be seen that the calcium-bearing lesions lie close to the trachea and bronchi, and always, in our cases other than fatal infantile disease, leave a clear space between the posterior margin of the calcium-bearing mass and the spine.

The contour of the calcium shadow is various; rounded, oval, irregularly nodular, pear-shaped. But in all instances verifiable from two angles, a contour, however hazy, is perceptible. The contour may, of course, be referable to lesions in one gland or in more than one in apposition (Fig. 7, to right of trachea), in a plane lying parallel to or at various angles to the incident ray. Occasionally it is desirable to make oblique exposures at various angles to see the full extent of large lesions situated one behind the other in the line of the incident ray.

The oblique position used is one very similar to the left anterior oblique of Pritchard.[8] A good view of the trachea and bronchi is obtained by using a position almost half way between the postero-anterior and the lateral. In the more nearly lateral positions there tends to be superposition of the two hilum shadows and the carina is not seen. In the oblique position the shadow of the aorta lies behind that of the spine. The trachea, carina and the full extent of the extrapulmonary bronchi are well seen. The patient stands with the head slightly inclined, the left arm hanging behind the left hip, the left anterolateral chest wall against the cassette changer, the right forearm bent over the head, the elbow as high as possible. The scapula thus interferes not at all with the view of the carina and right bronchus. Films taken in this position show many rounded and oval densities due to trunks axial to the incident ray. These are usually not confusing in the lower third over the heart shadow, but may be so just to the right of the right bronchus where it leaves the carina. These densities, however, rarely have the granular or stippled quality of calcium infiltration, and they change in quality and contour on slight rotation of the right side forward or backward. If these shadows due to vessels running to the apex prove confusing, the oblique exposures may be made with the right side closer to the cassette changer. The trunks then appear as curving or snarled ascending linear shadows.

Physical Signs. Our experience with physical signs in the diagnosis of tracheobronchial disease has been disappointing. Prolongation of the distinct whispered voice downward was common to the third, and not infrequent to the fifth dorsal spine; but we were unable to account for it on the basis suggested by d'Espine. The level to which it was heard often varied in the same individual, depending on whether the head were erect or slightly overextended, or bent forward, being lowest in the last position. Possibly this is due to some increased conductivity of the tissue between the trachea and the spine. But there was in our cases no definite relation of the sign with the presence of calcium-bearing tracheobronchial glands, and, as noted above, none such could be seen extending to the spine (see Figs. 7 and 8).

Percussion gave almost equally unsatisfactory results. There was no constant relation between apparent changes of percussion note and a demonstrable lesion.[9] It is not easy to understand how lesions other than the massive caseation of fatal infantile tuber-

culosis, which alone, apart from the malignant tumors, protrudes materially beyond the spinal margin, could supply an anatomic basis for changes in percussion. The width of the mediastinal shadow must be considered in relation to type of chest, fat, depth of respiration and cardiac phase. A mediastinal shadow extending uniformly 1 cm. beyond the spinal margin for the length of two posterior interspaces has been seen in the short, thick-set build at about the fifteenth year. We have seen essentially this same mediastinal outline both in normal noncontact children without a lesion and in a child with a calcium-bearing lesion 3 by 3.2 cm. lying behind the trachea and lapping for 1.4 cm. beyond the right tracheal wall. This child had been exposed to a source of infection for one year three years previously.

Nor is there commonly a well-defined basis for those changes in percussion in the interscapular region ascribed to changes in muscle tone. If such changes occur and be referred to reflex effects, one must usually assume that caseation within a gland is the cause. For while caseation involving the gland capsule and spreading to the tracheal wall is not uncommon enough to be a postmortem rarity, neither is it so frequent as to be invoked as explanation in the average clinical case.

Symptoms. We have not as yet been able to recognize a characteristic or definite group of symptoms due to tracheobronchial tuberculosis. For example, the child whose film is shown in Fig. 8 looked well and gave no complaints. Malnutrition and fatigue occur perhaps more often in affected children than in others in the same environmental and sociologic status. Respiratory symptoms have been lacking in cases of uncomplicated tracheobronchial disease. It is essential to rule out parenchymatous infiltration when cough or dyspnea is present. This requires, especially in young children, very careful technic. Neither in postmortem material nor in the living have we seen evidence that tuberculous tracheobronchial enlargement may give rise to mechanical stenosis. The areolar tissue about the tracheobronchial nodes and the parenchyma of the lung are very much more easily displaced than the air-passages. Unquestionably, extension of caseation through the gland capsule, if it involves the tracheal or bronchial wall, will cause respiratory symptoms. But in these cases it is reasonable to assign them to the concomitant inflammation. Even evidence of stenosis of branches of the vascular tree has been lacking. When

this diagnosis is suggested from the prominence of trunk markings arising near a calcium-bearing gland, it is well to look closely for infiltration in the lung supplied by the prominent branches. Decreased pulmonary elasticity, with consequent reduction of vibration set up by the pulse wave, is much more frequently a cause of locally prominent trunk markings than is thickening of the trunks, whether by disease of the wall or by increase of content. This, however, is a subject to be dealt with more fully elsewhere.[10]

Tuberculin Reaction. The quantitative tuberculin reaction is important at those ages at which it is most desirable to recognize the presence, pathologic condition and significance of tracheobronchial tuberculosis. A full statement of the value of the test is given elsewhere[1] but certain of its correlations which are pertinent in the consideration of tracheobronchial tuberculosis are cited here.

It has been found that, in the urban population studied, marked reactions with the lowest quantity used, 0.01 mg. O.T., are the rule at maturity, but that the younger the child the more often is such susceptibility a result of intimate contact with an open pulmonary tuberculosis. And of those children having the more severe reactions, almost two-thirds showed roentgenologic evidence of the primary complex, in large part lesions involving the glands. From the other point of view, considering the group with demonstrable tracheobronchial lesions, 90 per cent reacted with 0.01 mg. of tuberculin. These facts must be aligned with the evidence cited above, that only the presence of calcium infiltration allowed one to perceive the presence of glandular lesions, and with the observed interval of over three years that usually elapsed between the onset of intimate exposure to a source of tubercle bacilli and demonstrable calcium infiltration of the glands. (Calcium commonly appears earlier in a parenchymal lesion.) Therefore, in infants a tuberculin reaction, especially if intense, is in itself an indication for the institution of every measure calculated to improve the child's condition, and above all to prevent the lodgment of additional bacilli, whether a lesion can be shown or not. As stated above, tuberculous glandular enlargement sufficient to bulge beyond the spine shadow in infants is, in our experience, of fatal import. A working diagnosis and an urgent therapeutic indication is established by the tuberculin reaction alone at this age. A marked reaction to 0.01 mg. of tuberculin in a child under five years in still sufficient to warrant therapeutic and preventive means even in the absence of demonstra-

ble lesion. The presence of a recognizable lesion gravely emphasizes the danger and rarely before the fifth year will the calcium shadow be other than fine and presumably labile (Fig. 9). Even after the fifth year an intense reaction suggests an active lesion especially if the calcium infiltration casts a soft shadow.

The diagnosis in the individual of tuberculous tracheobronchial disease must rest on a demonstrable lesion, and a definite symptom complex can be discovered only by study of cases presenting such lesions. If diagnosis is to constitute a basis for adequate treatment, it is essential to seek out the infecting source. Intense tuberculin reactions in several children of the same household may point to an unsuspected or to an apparently arrested source of infection. The prevention of massive or prolonged reinfection is no less essential to the rational treatment of the tuberculosis of childhood than to any radical prophylaxis deserving the name. Comparisons of the incidence and intensity of tuberculin reactions and demonstrable lesions in contact and in noncontact families,[1] point to the paramount influence of contagion on the development of tuberculous disease in children. There is reason to believe that from the latent infections of individuals heavily infected in childhood develops the manifest tuberculosis of young adult life, economically the most wasteful form of the disease and, in its turn, the prolific source of latent disease in others.

Conclusions. 1. Study of postmortem material shows that calcium infiltration is the sole distinctive roentgenographic indication of the site of a lymph node situated within the limits of the mediastinum. Calcium-free glands, however enlarged, fail to cause perceptible intensification of the mediastinal shadow.

2. The protrusion of the mediastinal wall beyond the shadow of the spine and sternum by caseous lymph nodes is rare except in fatal infantile cases.

3. Intrapulmonary glands must contain calcium to be recorded roentgenographically. When they are large, that part of their calcium-free margins which projects beyond the arterial main stem will be recorded by contrast with the pulmonary parenchyma.

4. It appears that reabsorption of calcified caseous necrosis within lymph nodes does not occur.

5. Shadows simulating calcification are thrown by vessels axially radiated.

6. No symptom complex has been found characteristic of tracheo-

bronchial tuberculosis. Respiratory symptoms due to uncomplicated tracheobronchial disease do not occur.

7. The quantitative tuberculin reaction gives important indications as to the activity of the lesion in the individual. It is of unique value in infancy. It gives indispensable information regarding the source of infection, ignorance of which frustrates alike treatment and prophylaxis of tuberculous disease at its most tractable phase.

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DISCUSSION

Dr. Eugene L. Opie: It seems to me that in this study Dr. McPhedran has put the subject of tracheobronchial tuberculosis of children on a very definite basis. He has not depended for the diagnosis on thickening of the mediastinal shadow or on increased density in the hilum, for tuberculosis of tracheobronchial lymph nodes has been recognized in roentgenograms by changes identical with those seen in plates prepared from lungs excised postmortem. These changes are the result of calcification, but calcification has often just begun and does not indicate that healing has occurred. He has very clearly shown that the changes he has described are often associated with active disease. It is significant that almost all instances of tracheobronchial disease have been in families in which there has been a member, usually a parent, with open tuberculosis. In association with exposure to massive infection, apical tuberculosis has not infrequently developed. As in the film he has shown, apical disease is often on the opposite side from the tracheobronchial lesion and does not seem to be a

part of this first infection. It is apparently a superimposed exogenous infection occurring as a result of continued contact with an individual with open tuberculosis. These latent tracheobronchial lesions of children have grave significance and often suggest that manifest disease may follow.

Dr. Henry K. Pancoast: It has always been my impression that calcification implies a healing tuberculous process, and if the diagnosis is based on calcification alone, the case as seen at that time certainly cannot have a very active process. Is that not true?

Dr. Opie: It is true in adults, not in children.

Dr. Pancoast: In the study of several hundred cases by the Normal Chest Committee of the National Tuberculosis Association, of which Dr. Landis and I are members, calcified tracheobronchial lymph nodes were found in the great majority of adults. Dr. Opie has taught us all that the calcified nodule in the adult lung is of little or no consequence, but in the child it may be associated with an active lesion at the time. The only instances in which I have made a diagnosis of trachebronchial lymph-node tuberculosis were in cases in which very large glands were evident, and it seems hard to realize that small calcifications in the chest, without any apparent gland enlargement, must be regarded as evidences of an active tuberculous process. We do realize, however, that calcification is common in tuberculous glands elsewhere, as in the neck, and the presence of calcification by *x*-ray is regarded as diagnostic.

Dr. McPhedran (closing): I think the point is that these little round dots of calcification are not so significant as are the large granular deposits. The latter are found commonly in cases having manifest diffuse infiltration, and we have seen infiltrations develop in cases which at the first observation had these large granular calcifications alone. The determination as to whether the small dots are calcifications is essential pathogenetically. It may not be essential to identify them in the individual case.

The "broad mediastinum" is so often an artefact, in one picture you may see the mediastinum broad, in another narrow, depending on the depth of respiration. Broad mediastinum due to tracheobronchial tuberculosis did not occur in our cases except in infants in whom the disease had a fatal outcome.

THE ETIOLOGY AND PATHOGENESIS OF ACHYLIA GASTRICA*

The Nathan Lewis Hatfield Lecture
Lecture VII

By Prof. KNUD FABER
COPENHAGEN, DENMARK

By achylia gastrica is understood, as is well known, the very common morbid condition in which the stomach has lost its power to secrete gastric juice and especially acid gastric juice. The name, which was introduced by the American, Einhorn, in 1892, was given by him the special meaning that it was a lack of secretion which was not due to any atrophy of the gastric mucous membrane, but depended on certain nervous disturbances, therefore a *functional* and not an *organic* anomaly.[1] The phenomenon itself, the failure of the secretion, was previously known, as far back as Kussmaul and von Velden's time. It was first observed as a sign of cancer of the stomach and afterward also apart from this lesion, and in such cases it was interpreted as the result of atrophy of the mucous membrane, especially its glands, and as a sign of anadeny, that is the complete absence of glands. This was maintained particularly by Ewald and other German investigators, and it was claimed that this anadeny had been demonstrated anatomically. Einhorn, however, said that he had shown the presence of well-preserved gastric glands in cases of chronic achylia and that he had also seen chronic achylia cease after a certain time, in one case after some years, which was incompatible with the idea of anadeny.

While investigators were divided between these two views Martius proposed a third. He believed that Einhorn was right in holding

* Read February 12, 1926.

[1] It should be pointed out that achylia, strictly speaking, refers to an absence of gastric ferments as well as hydrochloric acid; but is here used, as is the general custom, to indicate absence of hydrochloric acid. With correct examination a trace of pepsin is very often found.

that the achylia was functional and occurred without atrophy. But he explained it not as a result of nervous disturbances but as a congenital anomaly, a constitutional weakness of the organ which could be hereditary and a family trait like other constitutional diseases. He called this form achylia gastrica simplex or constitutional achylia.

Against these three different views, and especially in opposition to Martius' conception, I have maintained:

I. That chronic achylia has an exogenous cause and is produced by external factors acting on the stomach either by direct irritation of the mucous membrane or through the blood circulation by a toxic action on the gastric parenchyma.

II. That the anatomical picture of gastritis will develop in both cases, and that the gastritis can lead to more or less atrophy of the glands; but that achylia occurs at such an early stage of the gastritis that there is no question of atrophy, to say nothing of anadeny. That achylia therefore may be found in combination with anatomically preserved glands and undamaged gland cells, although there is a well-pronounced gastritis.

III. That in gastritis we ought not to picture a superficial catarrhal inflammation, a stomach catarrh; but a disease of the glandular parenchyma in analogy with nephritis or hepatitis.

On inquiring what arguments can be advanced for and against these four different conceptions of the pathogenesis of achylia, and more especially what there is in favor of the interpretation of chronic achylia as the result of exogenous gastritis, we find that there are three lines of investigation to be probed: (1) Clinical experiences; (2) anatomical examinations; (3) experimental investigations.

Beginning with clinical investigations, there is first of all a phenomenon which formerly was considered very important, namely, that achylia is very often entirely symptomless, and is a chance finding when a large number of persons are examined. This was thought to be incompatible with the idea of a gastritis and a proof of a functional or constitutional origin. But it is easy to show that this argument carries little weight, because the best recognized exogenous achylia, alcoholic gastritis, especially in the later stages, is very commonly quite free from symptoms, and the same applies to the achylia in phthisis and other undoubted exogenous forms. Moreover this lack of symptoms is only temporary

in most cases; a careful examination will usually sooner or later disclose symptoms in the digestive organs.

A greater help in understanding the question can be obtained from an investigation into the etiology of the achylia. When one thinks of the extraordinary frequency with which achylia is encountered when a systematic examination of a large number of patients is made, one cannot but marvel how comparatively seldom it is that a definite etiology can be made out. The reason must be, first, that we are still in doubt as to what causal factors should be looked for; but, second, that when achylia is once established and has lasted a certain time, it will persist for the rest of the patient's life in a large number of cases. When one has to deal with a patient suffering from chronic achylia, therefore, one often has to go back to a much earlier period of the patient's life, perhaps back to the years of childhood, to find the cause of the disease. It is never possible to gauge the duration of the achylia from the length of time that the symptoms have been present, as it is so often entirely without symptoms.

The tendency of achylia to persist when once it is established makes it more common with increasing years. Thus Seidelin, in Copenhagen, found absent or greatly diminished secretion in 40 per cent of the working class above the age of fifty years. In old persons of sixty-seven to ninety-two years of age Seidelin, in Oslo, found anacidity in 67 per cent.

It is consequently much more important to study the occurrence of achylia in *childhood;* it will probably be most easy to detect its cause then. It is also important for another reason. In support of the view that achylia is a constitutional disease it has been said that even in small children achylia could be demonstrated, and this was taken as proof that it was congenital. A careful study of the gastric secretion, especially the achylia of infants, has been made by different Danish clinicians (Meyer, Jacobsen, Hertz, Andersen and particularly by Chiewitz and others in C. E. Bloch's pediatric clinic in Copenhagen). In later years similar investigations have been undertaken in clinics elsewhere especially in America, for example by Marriott in St. Louis, in New Haven, and so forth. By investigations of the gastric secretion in small children it has been found that it is best to avoid milk as a test on account of its large content of "buffer substances," especially phosphates and calcium caseinate, and this applies particularly to cow's milk. Meyer's test meal of

barley water, which is most satisfactory, has proved that achylia can fairly often be demonstrated in small children, but that very obvious causes can usually be detected to account for it, and that it is most often transitory. Chiewitz and Andersen found in 127 children nothing which could be regarded as congenital achylia, and a number of other investigators have come to the same conclusion. The causes of achylia which can be detected in young children are chiefly two—parenteral febrile diseases and affections of the gastro-intestinal canal, particularly the varieties of infectious enteritis.

The various febrile diseases affect the gastric secretion in small children by diminishing the production of acid. This appears not only during the febrile stage itself, under the influence of the increased temperature, but as hypochylia or achylia occurring in convalescence and often lasting a long time. Hertz, Chiewitz and Andersen observed this, for instance, after influenza, angina, pneumonia, bronchitis and similar complaints. In some cases a normal acid secretion was demonstrated in the children before the acute disease started and after it was over there was an achylia of long duration.

Far more commonly, however, was achylia detected in small children with gastroenteritis. The stomach seems indeed to be influenced very easily by infectious enteritis and chronic achylia is often found in such children. It may disappear in company with the intestinal disturbance, or may persist for a long while. It may be attended by anemia and marasmus, or it may occur alone. In 33 out of 64 small children with chronic gastrointestinal dyspepsia and atrophy, C. E. Bloch found a diminished secretion of acid at the climax of the disease. In 18 cases the secretion rapidly returned to normal at the same time that a general improvement took place, but in 15 cases there was complete achylia, lasting for weeks or months, observed up to eleven months. In some of these also it could be shown that the secretion became normal again; in other cases the achylia seemed to be lasting and associated with profound damage to the secretory apparatus.

In opposition to the theory that the achylia is congenital, we find, then, well-marked exogenous achylia and obvious causative factors. The latter chiefly consist of acute infections, especially of the intestinal canal. It is important to note that achylia in children is often a temporary phenomenon, more frequently than in adults, but that, on the other hand, it may last a very long time. In

certain cases it seems to last years without hindering the nutrition and fairly normal development of the child.

Passing on now to *adults*, we observe quite analogous conditions, and there is cause to dwell particularly on the cases of achylia in the different groups where an exogenous origin can definitely be detected.

A very obvious cause is the direct damage and irritation of the mucous membrane of the stomach from taking alcohol. Vogelius has investigated a large number of patients in Copenhagen with chronic alcoholism. He could demonstrate achylia in 50 to 60 per cent. What is of special interest here is that he could show that some of the patients who came into the hospital with signs of recrudescence of the gastritis, namely, with severe dyspeptic symptoms, vomiting and so forth, had achylia; but that it disappeared fairly rapidly when the patient abstained from alcohol and was on a light diet. Not before these bouts had been repeated several times did the achylia become constant. The lesion of the gastric mucous membrane had increased and the failure of the secretion was permanent.

In contrast to this direct action on the mucous membrane, there is the change which occurs during a *general intoxication* of the organism and above all in acute infections. Just as in children, it is especially the intestinal infections which affect the stomach, as for instance, typhoid fever, paratyphoid fever and dysentery. Achylia is very commonly found after these diseases, particularly the last, and just as in the case of children, it may disappear again in the course of some weeks or months. In a certain number of cases, however, the achylia remains, presumably for the rest of the patient's life. We see the same thing in chronic infections, especially tuberculosis. In phthisis Permin has demonstrated achylia in 34 per cent in the early stages, and even in 75 per cent in the final stage of the disease. An intoxication is the most natural explanation. Guido has recently discovered achylia in 26 per cent of malaria cases, and in some cases the achylia has been constant. In syphilis, achylia also is common. Apparently, widely different infectious diseases are able to influence the gastric secretion in a similar manner.

But it is not only the intoxication produced by infectious diseases that can give rise to achylia. We have a striking example in Graves' disease, where it is very common and occurs in 50 per cent of the

patients. In this case it is also a question of an intoxication of the organism, but it is an autointoxication not caused by microbes.

The same applies to the achylia so frequently seen during pregnancy, especially in those cases of the intoxication of pregnancy which lead to eclampsia and similar serious conditions.

In Denmark, Kramer Petersen has shown that a diminished gastric secretion occurs in a great number of pregnant women, (in 86 per cent.) the last few months before labor and that an achylia is very common at this time. In patients with eclampsia, it can be demonstrated in 43 per cent of the cases.

We see, therefore, that the gastric mucous membrane and its secretory apparatus behave in a manner similar to that of other parenchymatous organs, especially the kidneys and liver, and that it is affected by infections and by intoxications. We must call these causes exogenous, and they are observed so frequently and are of such different kinds that they can fully explain the many cases where the cause of the achylia cannot be detected directly.

One of Einhorn's main arguments in favor of achylia in many cases being necessarily of functional origin was that the achylia might occur for a time and then subside. Such transient achylia is certainly inconsistent with the idea of a total atrophy of the mucous membrane, but it was wrongly interpreted as an argument against there being any inflammation whatever and as evidence against an exogenous origin for the achylia.

We have, however, in cases of alcoholic gastritis seen that the very common phenomenon occurs that achylia only remains for a time and then disappears when the abuse of alcohol is prevented, again to make an appearance when an exacerbation of the gastritis sets in. Likewise we have observed it to be very common in infants, that when their gastric secretion was reduced or suspended on account of some toxic influence, the secretion recurred after a longer or shorter period. It would appear as if the gastric secretion were much more easily influenced in children than in adults, is subsiding more easily and reappearing more quickly. In adults it is, however, a fairly frequent phenomenon that achylia may also disappear without it being possible for us to demonstrate such a direct irritant as alcohol. Patients with achylia come for treatment, but gradually as their condition improves by means of dieting and rest, the secretion recommences and after a period of hypoachylia we find normal figures at our examinations. Naturally this

is especially observed in cases of what might be termed pseudo-achylia, where by fractional examination a small and retarded secretion can be demonstrated, but it is also complete anacidity. The explanation of the fact that an achylia subsides and secretion returns is naturally to be sought in the improvement in the gastritis. In any case it may not be utilized as an argument against the existence of a gastritis. The younger the patients, the more frequently we note the return of the secretion; the older the patients and the longer the gastritis has persisted, the more stable is the achylia.

These conditions are further elucidated when we turn to an investigation into the *anatomical findings*. In chronic achylia we discover that it is a field which has only been thoroughly investigated comparatively recently. The investigations into the morbid anatomy of diseases of the different organs, which were so extensively carried out in the last century, usually did not deal with the stomach and intestines when it was a question of microscopic examination. The reason was principally that after death their mucous membranes are so quickly acted upon by the digestive fluids and cadaverous putrefaction. Hayem, in France, was the first to attempt to combat this by fixation directly after death. In conjunction with C. E. Bloch in 1899 I introduced the injection of a 10 per cent formalin solution (*i. e.*, 4 per cent formaldehyde) into the abdomen immediately after death. We thus obtained excellently fixed stomachs and could subject them to a thorough anatomical examination. I will show you a series of pictures of the changes seen in the gastric mucous membrane in chronic achylia. In many of the patients the digestive disturbances gave rise to extremely few symptoms or none whatever, and the cause of the disease was unknown. The material thus comprises those cases which from the clinical picture one would be inclined to regard as constitutional or nervous achylia, as cases of *achylia simplex*.

The first difficulty was to find really normal stomachs. It is not sufficient that there has been no morbid symptoms and that the secretion was normal. As in all other organs there is no absolute parallelism between anatomical lesion and disturbance of physiologic function, and a normal secretion may well be associated with anatomical signs of inflammation.

The first picture shows an absolutely sound stomach in a child who died from meningitis (Fig. 1). The glands are observed to be placed close together and there is no trace of interstitial inflam-

mation, no round cells between the gland tubules. In the following pictures, on the other hand, we see inflammatory processes throughout the mucous membrane (Figs. 2 and 3). The latter, however, may be so well preserved at first that the glandular layer is practically uninjured.

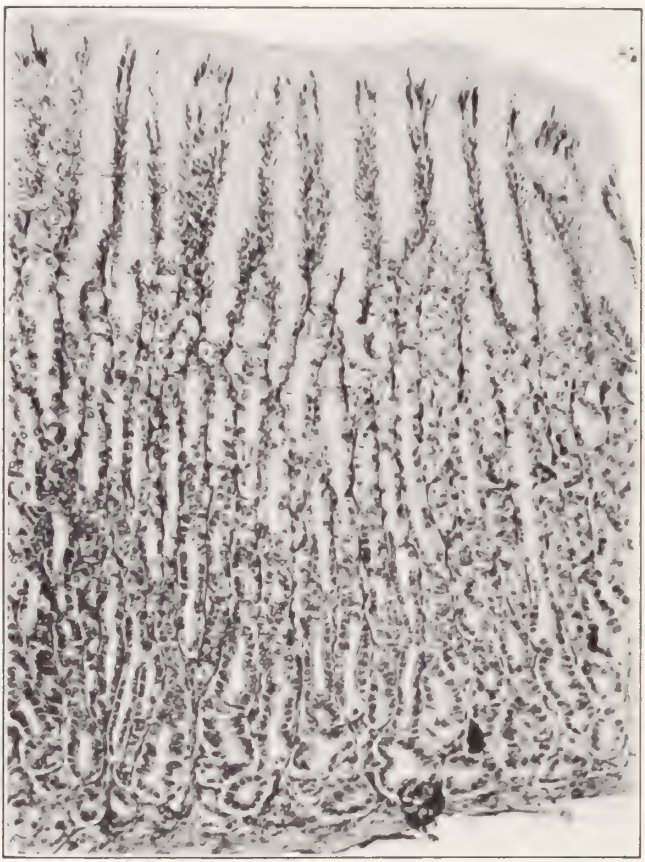

FIG. 1.—Normal mucous membrane of the stomach from child with meningitis.

In some cases the inflammatory phenomena are very strongly marked, but in others, where the achylia has only existed for a short period, only slight changes are observed. As an example, Fig. 4 shows the stomach from a child aged one and a half years. After being quite healthy for one year it got an acute attack of enteritis,

followed by achylia ascertained to have existed for six months, as the child died from other causes. The mucous membrane was well preserved, but a slight infiltration of round cells is observed. It is obvious that in this case the intoxication has directly affected the secretory apparatus and caused beginning anatomic changes.

FIG. 2.—Gastritis with achylia in a girl aged nineteen years.

Gradually as the inflammation develops signs of lesion of the glands put in an appearance. They get fewer in numbers, become separated from one another by the inflammation, atrophy, tend more and more to become vestiges and finally completely vanish (Figs. 5, 6, 7). We then see the stomach consisting only of granulation

tissue covered merely by the surface epithelium of the stomach. There is now complete anadeny. Other changes are also observed. The accumulations of round cells resembling follicles may have developed into large typical lymphoid follicles; follicular gastritis is present (Fig. 8). Cystic formations in the necrosing glands are

FIG. 3.—Gastritis with achylia in a man aged forty years.

often seen, and cysts may sometimes be very prominent. It frequently shows the curious phenomenon that the stomach's superficial epithelium is transformed into intestinal epithelium which is specially characterized by the goblet cells containing mucus. This is never found in the normal stomach. By means of a very thorough examination of twelve normal or almost normal stomachs, Lange,

in my clinic found normal gastric epithelium throughout, while in 14 stomachs in patients with achylia and gastritis, intestinal elements were discovered scattered about in 10 of them, that is, in 75 per cent. In the picture (Fig. 9) one sees Lieberkühn's glands with Paneth cells and only small nests of stomach glands. It seems as if these heterotopic formations especially occur when the gastric secretion has ceased, and the mucous membrane, therefore, practically functions as an intestinal mucous membrane.

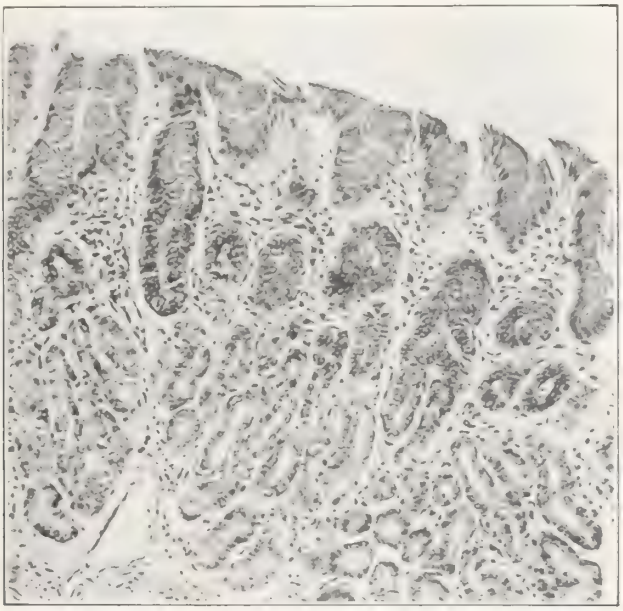

FIG. 4.—Achylia in a child aged one and a half years. Commencing gastritis.

Thus we see an even transition from the slightly affected stomachs to cases where there are severe antomical changes with profound injury to the secretory apparatus. Not until the gastritis has persisted for a considerable time do we arrive at the atrophic stage. The examples of complete atrophy I have shown you were found in aged or middle-aged persons, where the achylia may be presumed to have existed for many years. It is easily comprehended that there is achylia in these cases; it has, on the contrary, in agreement with Einhorn been emphasized that when the glands in the milder cases were well preserved, then the local affection of the mucous mem-

brane could not be the cause of achylia. As already mentioned the anatomical changes are but the signs of the disease visible to us.

An intoxication may influence the cells and their innervation apparatus immediately, even before anatomical changes have manifested themselves, and even in pronounced anatomical inflammatory

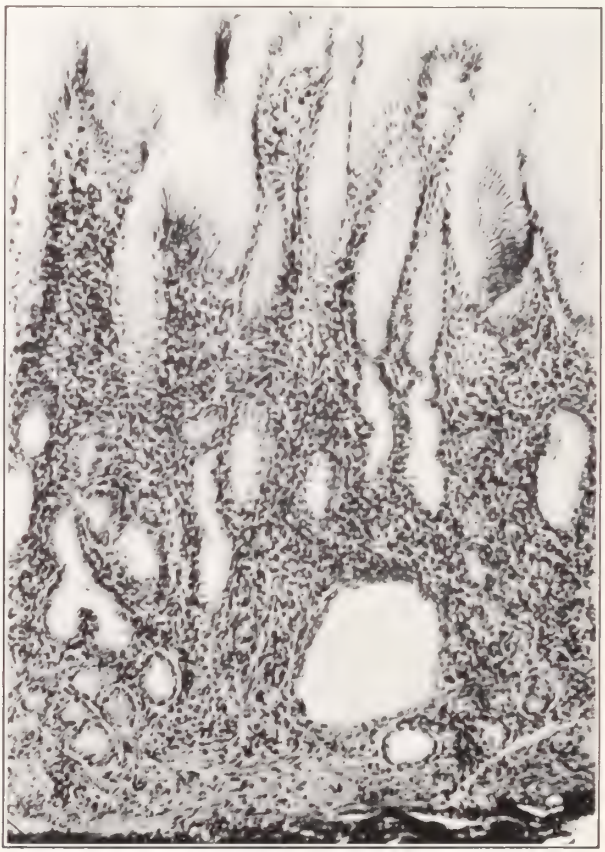

FIG. 5.—Gastritis alcoholica with achylia gastrica in a man aged fifty-one years. Commencing atrophy of the glands and some cysts.

phenomena the secretory cells may appear to be quite natural at the first anatomical examination. The accompanying Figure 10 is taken from a patient suffering from cancer of the stomach, who had showed symptoms for half a year before operation and who had a complete anacidity. By Altmann's stain we observe both the chief

cells and especially the acid-secreting cells to be absolutely normal and prepared to resume their secretion should the harmful influence subside. The next illustration (Fig. 11) originates from another patient with cancer ventriculi and achylia. Here the cells have commenced to degenerate and presumably will no more be able to resume their function.

That achylia in cancer is neither neurogenic nor constitutional all are certainly agreed. The pictures thus show clearly that we cannot conclude from the cells being preserved in a functionally active state that it is not a question of a local affection of the stomach, of gastritis.

FIG. 6.—Complete atrophy of the mucous membrane of stomach. No secreting glands. From a man aged forty-two years, who had gastric symptoms for ten years.

What now can we conclude from these anatomical investigations? We may infer that gastritis is a very common phenomenon, and that from an anatomical point of view it shows great similarity to chronic inflammation in other large glandular organs like the kidneys and liver. We find failure of the secretion as a perfectly natural result of the inflammation and atrophy of the glands, just as we find insufficiency of the organs of the kidneys and liver. We realize that we cannot look upon gastritis from the old standpoint, gastric catarrh. It is a disease of the whole gastric parenchyma, not merely of the mucous-producing top layer of the mucous membrane.

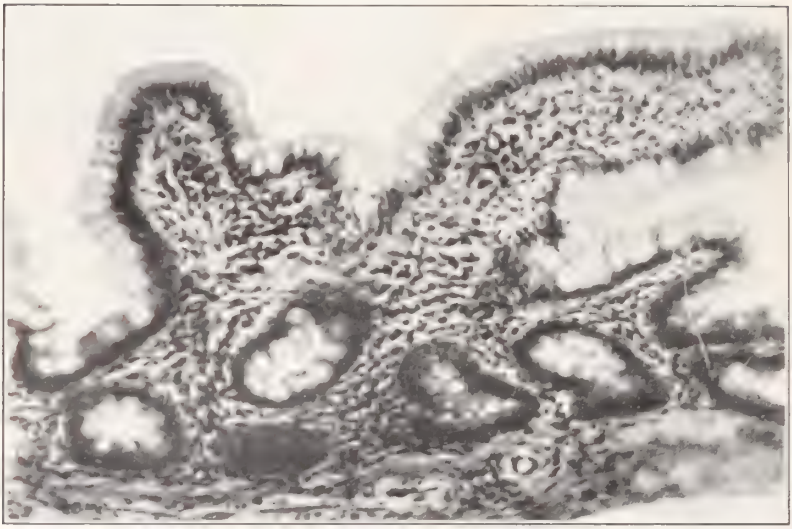

FIG. 7.—Atrophy of the mucous membrane of the stomach; complete anadeny. Gastric epithelium replaced by intestinal epithelium with goblet cells.

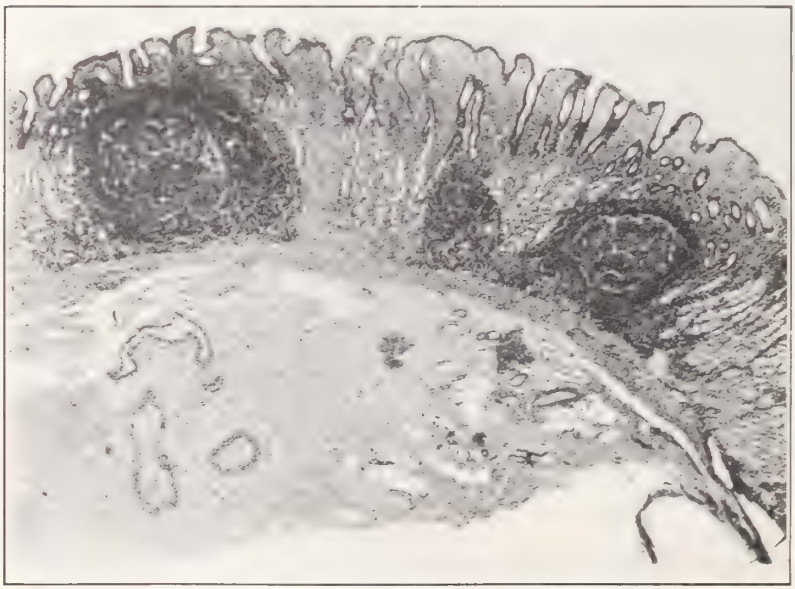

FIG. 8.—Follicular gastritis with achylia in a girl aged twenty-five years.

We understand that gastritis can be produced both by surface irritation and by hematogenous intoxication. We understand also that gastritis may occur which does not lead to achylia, as is frequently observed to a greater or less extent in gastric ulcer.

But does this imply that all achylia is always due to gastritis? Cannot the other causes — nervous influences or congenital anomalies — in some cases be the prime ones and the gastritis only a secondary effect, perhaps produced by the deficient gastric digestion.

FIG. 9.—Chronic gastritis alcoholica in an old man aged fifty years. In the mucous membrane nearly all the gastric glands have disappeared and are replaced by Lieberkün's glands with Tanable cells in the bottom. The epithelium is of the intestinal type with goblet cells.

If we accept the views given here about intoxication and direct irritation being the principal causes, we need no other actual explanation; but we cannot exclude, clinically or anatomically, that at all events in some cases the pathogenesis may be different from what we have put forward.

With regard to nervous achylia, its existence can hardly be denied, especially since Bennet and Dodds have produced it experimentally by suggestion under hypnosis. Another question is whether we can get genuine chronic achylia through nervous channels or only merely a purely temporary disorder.

About these questions we can obtain important information from the third group of investigations, namely, *experiments on animals*. We know from the work of Pawlow and his pupils and from Carlson and Ivy that in the secretion of gastric juice there is a primary cephalic period governed by an influence of a cerebral nature, induced

FIG. 10.—Gastric gland with well-preserved acid cells in a case of stomach cancer which had given symptoms for a half year. Complete anacidity. Altmann's stain.

reflexly or psychically through the vagus and a second humoral phase governed by chemical agencies from the blood. Not only a secretion but also an inhibition can in the first phase be produced through the nerve. After complete division of the vagi the secretion is observed to decrease markedly or entirely cease for a while. But this phase proves to be temporary, the secretion returning after

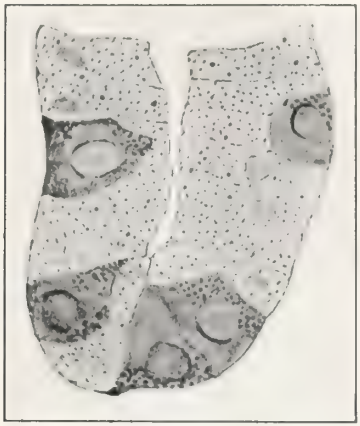

FIG. 11.—Gastric gland from a case of stomach cancer which had given symptoms for three months. Complete anacidity. The acid cells and the chief cells show degeneration. Altmann's stain.

FIG. 12.—Part of gastric gland in anacidity after cutting both vagi in a dog.

some time has elapsed. Even if the stomach is completely isolated from all its innervation, both sympathetic and parasympathetic, it yields a fairly abundant secretion after the introduction of food. The second phase can only be inhibited temporarily through nervous channels.

In accordance with this we do not see permanent but only temporary achylia of nervous origin. It is found moreover, when the gland cells are examined histologically, that in spite of the temporary failure of the secretion, they preserve their appearance as in actively secreting glands. They are ready to resume their work at any time whatever, just as we have seen it in the beginning gastritis.

I can illustrate this with pictures for which I am indebted to an interesting work by Einar Thomsen carried out in the Physiological Institute in Copenhagen. In dogs with a gastric fistula, he was able to confirm that an achylia could be the result of cutting both vagi and that the completely nerveless stomach was still capable of secreting. Only transient achylia could be provoked through nervous paths, and the anatomical examination of the secreting cells showed normal appearance (Fig. 12). On the other hand, he could produce a true chronic achylia experimentally by the other methods we arrived at from a consideration of the clinical factors, namely, by direct irritation of the mucous membrane, or through the blood, by bacterial intoxication. Gastritis was caused by the administration of alcohol which first manifested itself by transient hypersecretion and then by more or less complete achylia. Anatomically there were signs of gastritis with interstitial changes as in man, but the gland cells ceased to secrete long before they began to degenerate.

For bacterial toxin he used chiefly diphtheria toxin. By the intravenous injection of this poison a very pronounced acute gastritis with great hyperemia and interstitial changes were produced just as we saw in the picture I have shown of human gastritis. The secretion ceased and here he could rather soon see the degeneration of the glandular cells (Fig. 13).

Thomsen could also produce a pure toxic diminution of the gastric secretion by feeding his dogs with thyroid extract. The gastric secretion nearly ceased but no real achylia was produced and the secretion returned when the thyroid intoxication was stopped. In this case there was no sign of gastritis, during the experiment.

These experimental researches thus prove to us that we may sometimes see a passing achylia of nervous origin, but that a chronic

achylia arises either by direct injury to the stomach or by hematogenic intoxication. This agrees with the clinical and anatomical studies.

It remains to say a few words about the theory advanced especially by Martius of the constitutional achylia, the achylia gastrica simplex. He believed it was due to a congenital weakness, and that it was a true constitutional disease. There is no doubt that he

FIG. 13.—Gastric gland with degeneration of both sets of cells in gastritis after poisoning by diphtheric toxin.

came to that conclusion because achylia was so common and so often symptomless. I have shown that these arguments are without value. But an attempt has been made to support the hypothesis: (1) By showing that achylia can occur in children, in fact, even in small children, and (2) by exploiting the fact that it may occur as a family disease which attacks several members of the same family.

I have already made clear the situation with regard to the children and have shown that no argument is forthcoming from this quarter

in favor of the theory of congenital achylia. It would be of greater value if the occurrence in families could be demonstrated on a larger scale. It would be necessary, however, to be rather cautious, as achylia is so extremely common a disease. Large and convincing series of cases in families I have not yet come across. The frequent occurrence of achylia among brothers and sisters or relatives would not in itself put an exogenous origin out of court, for example, an intoxication by infection. It would at most show a greater family susceptibility to external influences, which must be distinguished from a genuine congenital hereditary disease, as, for example, Huntington's chorea.

Summary. We have arrived at the result, then, that the very common and very important disease which we usually call achylia gastrica has nothing particularly remarkable in its etiology and pathogenesis which marks it out from the diseases of other organs. It is due to disease of the gastric mucous membrane with its extensive glandular apparatus. When it is excited through nervous channels it can only persist for a short time, but apart from this it is due to the same causes which we know produce disease in other organs direct irritation or hematogenous intoxication by bacterial toxins or autotoxins. With the exception, perhaps, of the initial stage of intoxication, the natural term for the disease is gastritis, by which is to be understood, not the old conception gastric catarrh, but a disease of the glandular parenchyma of an inflammatory nature.

The reason that the anatomical side of the question has come under consideration much later in this case than in that of other glandular organs like the kidneys and liver, is because postmortem changes take place so rapidly. As soon as one has learned how to avoid these, the analogy is obvious and gastritis takes its natural place by the side of nephritis and hepatitis.

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HEPATITIS AND ITS RELATION TO CHOLECYSTITIS*

The Mütter Lecture

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In several papers written during the last nine years, Evarts Graham,[1,2,3] whom I think your committee honored the year before last as it has me this year, has presented some interesting observations and stimulating suggestions on the pathogenesis of hepatitis and cholecystitis, on the path of infection in diseases of the gall-bladder and on the association of cholecystitis and hepatitis.

In reading these papers and others on the same subject, I am reminded of a saying of the hematologist, Naegeli, quoted by Aschoff.[4] He writes: "It is a sure sign of the undeveloped state of science if the logical concepts with which it deals are not fixed and when multiple meanings are assigned to the same term." I am reminded of this saying because of the confusion in my own mind regarding the exact meaning of hepatitis and cholecystitis. Perhaps the disagreement and controversies that arise between clinical surgeons, pathologists and physicians, when terms such as hepatitis and cholecystitis are used, are inevitable in the uncertain state of much of our knowledge regarding these subjects and the different conceptions formed when these terms are used.

At the outset it may be well to refer briefly to the observations that have been made regarding hepatitis and cholecystitis by a number of surgeons during the last ten years and to state the conclusions that have been drawn from these observations. Small sections of the liver removed during operations for cholecystitis have regularly shown varying degrees of hepatitis. The lesions are

* Read, December 10, 1926.

confined largely to the interstitial tissue where the lymphatics are situated. In several instances pieces of liver removed during operations for chronic appendicitis have shown inflammatory changes. Gall-bladders removed for a group of clinical symptoms which are classed as dyspepsias, and in which there was no history of gall-stone colic, have not infrequently shown very slight or no macroscopic lesions, but have shown, under the microscope, slight inflammatory infiltration of the deeper layers of the walls of the gall-bladder. The conclusion has been reached that in many cases of cholecystitis there has been a direct extension to the wall of the gall-bladder from an inflamed liver; that frequently a vicious circle exists between the gall-bladder and the liver whereby each may infect the other; that the infection of the liver from an inflamed gall-bladder is an important factor in the production of cirrhosis; that lesions of the gall-bladder wall that cannot be readily recognized macroscopically give definite clinical symptoms.

I may also add at the outset that anyone having the temerity to discuss hepatitis is thrown on the horns of a dilemma. Either he must discuss the subject as if hepatitis were a simple entity or he must refer to its very complex and diversified etiology and be carried into a rather loose-jointed and rambling discussion. I must beg your indulgence for the latter method.

I have thought, however, it might be of advantage to bring forward some of the problems that present themselves when the somewhat indefinite word hepatitis is used and to consider the interesting question as to when the reaction of the cells that make up the liver and the gall-bladder reaches the plane of clinical observation; in other words, what degrees of reaction in the hepatic cells or the gall-bladder wall are we justified in considering clinical entities?

There are a few features in the microscopic anatomy and physiology which seem to me pertinent to the discussion. The arrangement and relation of the blood and lymph capillaries to the liver cells are imperfectly understood. There is, however, a fairly general agreement that the portal vein and the hepatic artery spread out into the intricate capillary network, which is very intimately related to the liver cells. The Danish zoölogist, Krogh,[2] who has made most extensive studies of the anatomy and physiology of the capillaries, considers that the endothelium of the hepatic capillaries is a syncytium with numerous nuclei but without defined cell borders as in the embryonal capillaries, and that the star cells of von Kupffer,

which appear at rather short, regular intervals, are an integral part of the capillary wall. The English histologist, Schafer,[9] believes that what remains of the endothelium of the liver sinuses is represented by these stellate cells.

Blood, entering the liver through the portal vein and hepatic artery, spreads out in a large and complex capillary network, the liver cells being directly bathed by blood and not by lymph, as elsewhere in the body. Besides the polygonal liver cells and the von Kupffer cells which make up, with the capillaries, the lobules, there is a connective-tissue stroma underneath the serous covering and between the lobules which supports the bile ducts and blood-vessels in their course through the organs. The lymphatic vessels arise from the lymph capillaries in this connective-tissue stroma. Numerous lymph vessels accompany the interlobular branches of the portal vein and numerous lymph vessels accompany the hepatic vein.

From the study of the injection specimens made by Sappey,[7] it seems that a considerable number of fairly large lymph vessels pass along the inferior surface of the liver over the body of the gall-bladder from the adjoining quadrate lobe and the right lobe, to form an extensive anastomosing network of vessels in the neighborhood of the neck of the gall-bladder. Others, derived from the same sources, pass behind the gall-bladder to terminate in the same region. There are apparently numerous anastomosing lymph capillaries between the lymphatics of the liver and the gall-bladder.

The hepatic cells have many and diversified functions. The liver is the great laboratory and storehouse of the body. It is engaged in chemical transformations, demonstrated by the high consumption of oxygen and the production of heat. All three classes of foodstuffs -carbohydrates, fats, and probably proteins are built up into large nondiffusible molecules and stored in the liver cells and, by reversible ferments, again changed into soluble forms which pass out into the circulation as needed. The liver cells change the amino-acids to simpler compounds, produce bile with its well-known function and play an important part in the coagulation of the blood.

But is is to a function which it shares with the reticulo-endothelial system, in rendering harmless various toxic substances and in the destruction and disposal of living microörganisms and red blood cells, that I wish to call special attention. The stellate cells of

Kupffer, which we have just said are part of the lining endothelium of the liver capillaries, belong to the group of reticulo-endothelial cells which are found in the sinuses and pulp cords of the lymph nodes, the reticulum and the blood sinuses of the spleen and the capillaries of the bone-marrow. These cells behave in a special way in taking up dyes and other substances and show a pronounced capacity for phagocytosis.

Kupffer showed that the star cells in the liver were loaded with the pigment after the injection of india ink. These experiments have been many times confirmed. Various chemicals injected into the circulation are taken up in the same way by the stellate cells. Certain salts are not arrested; the cells show a power of selection and certain substances are taken up and stored in the liver cells. A number of careful experiments by Roger[8] and others have shown that alkaloids are arrested in the same way in the liver. Some of the alkaloids lose a portion of their toxicity in passing through the liver; some of them are excreted in the bile and, according to Roger and others, the detoxicating effect varies with the abundance of glycogen in the liver.[9] There can be no question of the arrest, storage and destruction in the liver of a number of simple and complex chemical substances.

The action of the liver on microbes is equally interesting. Wyssokowitsch[10] showed, many years ago, that pathogenic and non-pathogenic microbes and the spores of molds were arrested and taken up by the cells of the capillaries and sinuses in the liver, bone-marrow and spleen. The capillaries and sinuses in these regions are great settling basins, where all manner of minute foreign substances are deposited and taken up. Although all the cells of the so-called reticulo-endothelial system in these situations are largely concerned in the reactions, it by no means implies that their behavior in the liver, spleen and bone-marrow is the same. The stellate cells of the liver are not the exact equivalent of the endothelial cells of the splenic sinuses, for example. Bacteria are not taken up to the same extent in all the depots of this system of cells. The Kupffer cells of the liver seem to take up and retain large numbers of microbes at an early stage, at least in inoculation with certain microbes and in certain animals.

Bacteria and other foreign substances taken up by the reticulo-endothelial cells in the bone-marrow and spleen are destroyed there or start to grow and invade again the blood stream. But for the

material deposited and taken up by the liver cells there is another possibility. The foreign particles may be excreted in the bile. In Wyssokowitsch's (*loc. cit.*) experiments he expressly states that the bacteria taken up from the blood stream do not pass out by the excretions and secretions of the body. But it has been proved over and over again, during the last forty years since he made his precise and interesting observations, that bacteria injected into the portal vein or the systemic veins, if injected in sufficient dosage, regularly enter the bile. Nichols[11] recorded in 1916 a number of carefully carried-out experiments and reviewed and criticized the work done up to that time by Fütterer, Chiarolanza and Koch. He injected varying doses of typhoid bacilli and cholera vibrios into the systemic veins and into the portal vein in immune animals and in animals in which no immunity had been established. He found fairly large doses were necessary to make bacilli appear in the bile, and a larger dosage was required in the systemic veins than in the branches of the portal vein. He records several instances in which the results were strikingly inconstant; the same dosage injected in a portal vein in one instance showing numerous colonies cultured from the bile, and in another showing not a single colony. There were marked individual variations in elimination. Immune animals showed a greater power of excreting bacteria in the bile than nonimmune animals. Rapid agglutination *in vivo*, deposition in the liver and corresponding elimination are suggested as explanations. The bacilli began to appear in the bile with astonishing rapidity, being found in two to three minutes after portal injection, and the first plates showed the largest number of colonies.

The reticulo-endothelial cells are concerned not only in taking up foreign bodies and, through the action of various intracellular ferments, modifying or destroying them, but they are also concerned in furnishing and secreting various substances which aid in the destruction of microörganisms and in the neutralization of their toxins. Metchnikoff[12] wrote that it was probable that the macrophages (and in his macrophage system is found the first suggestion of a reticulo-endothelial system) represented the principal source of antitoxin. Since then it has been shown by Hahn, von Skramlik and Huenermann[13] that the perfusion of the liver of a sensitized animal absorbs the corresponding antigen. The stellate cells of Kupffer are assumed to be the active agents in the liver. Ehrlich and Morgenroth[14] observed that in dogs poisoned by phosphorus,

with a consequent degeneration of the liver, there was a diminished production of alexin or complement. Although later experiments have not by any means settled these questions, there can be no doubt that the reticulo-endothelial system, and consequently the stellate cells of the liver, play an important part in the taking up and destruction of bacteria and in the altered reaction of the body to introduced bacteria.

If next we turn to the circumstances under which bacteria invade the tissues and settle in the liver, and consider that, aside from the fact that in very many infections where organisms are introduced through the skin or through the genito-urinary system, the respiratory system, the mouth, the pharynx and esophagus, and in which bacteria gain entrance into the systemic circulation and are widely distributed, there is a large group where invasion occurs through the portal system.

Numerous observers have studied the intestinal flora. We all know that multitudes of bacteria, aërobic and anaërobic, inhabit and flourish in the large intestine and the lower portion of the small intestine, becoming less and less under normal conditions as the upper jejunum is reached. The intact epithelial cells, the protective mucus, the continual movement onward of the intestinal contents, all prevent the passage of bacteria through the intestinal wall. Slight alterations, such as congestion or catarrhal inflammation and demonstrable morphologic lesions may, of course, alter conditions so that the intestinal wall is penetrated. Fifty years ago Pasteur and his collaborators showed that the anthrax bacillus, which is so fatal for mice and guinea-pigs, could be swallowed with impunity. To produce anthrax invasion by the path of the intestine it was necessary to feed the animals powdered glass and sand.

I wish, however, to call your attention to a number of experiments which show that bacteria can pass through the normal intestinal wall. There is mention by Metchnikoff[15] of an unpublished paper by Mitchell on experiments carried out in his laboratory, in which animals were killed by the invasion of anthrax bacilli through the intestinal mucosa, to all appearances normal and in which the animals received no nourishment capable of producing lesions of the intestinal wall. The animals were fed anthrax spores mixed with a pap of bread soaked in milk. In 1894, Macallum,[16] in studying the absorption of iron, showed that leukocytes could pass out into the intestinal lumen, take up foreign material and return

through the intestinal wall. He recovered the leukocytes containing stained granules in the liver and spleen. The leukocytes found their way into both the systemic and portal circulation. The next year, Desoubry and Porcher[17] working in Nocard's laboratory, established by numerous experiments that during digestion bacteria of all kinds may pass through the normal mucous membrane of the intestines and may be found during several hours in the chyle and blood. These experiments were made at the suggestion of Nocard, who had noticed that serum obtained from blood withdrawn with every possible care from horses shortly after feeding was, every now and again, contaminated.

In studying the pathogenesis of tuberculosis, a number of experimenters have confirmed these observations. During digestion, many bacteria pass through the intestinal mucosa with the chyle and are found in the mesenteric glands, in the lymph of the thoracic duct, in the blood current and in the liver, spleen and bone-marrow. The younger the animal the more constant the results. The bacteria find their way through the mucous membrane of the intestinal wall without leaving a trace of their passage. Calmette and van Steenberghe[18] studied the mechanism of passage. They confirm the observations of Macallum. Polymorphonuclear leukocytes in large numbers pass through the mucosa into the lumen of the bowel normally. They form, with the epithelial débris, a not inconsiderable part of the feces. Attracted by chemotaxis to the surface of the intestine and the secreting surfaces of the glands, hundreds of thousands pass out daily. Bacteria in the intestinal lumen are taken up by some of these phagocytic cells. During digestion, especially when bacteria are intimately mixed with the food, the bacteria are carried by the leukocytes back through the intestinal wall to enter with the minute globules of fat or the salts of fatty acids, made by breaking up the fats, into the lymph vessels of the villi of the small intestine and hence to the systemic circulation, or they find their way, to a less extent, into the capillaries of the villus and to the portal circulation.

The bacteria taken in may be destroyed by the intracellular ferments of the leukocytes or the leukocyte-containing microbes may be arrested and destroyed in the submucous lymphatics or in the lymph nodes. However, not infrequently, living bacteria reach the liver and are destroyed there. Ford[19] showed that 70 per cent of the livers removed aseptically a minute or two after death yielded

cultures of microbes similar to those found in the intestine. The bacteria seemed to be feeble and attenuated and were slow to grow. The presence of tetanus antitoxin in the serum of individuals who carry tetanus bacilli in the digestive tract has been shown by Ten Broeck and Bauer.[20] They showed spores of tetanus bacilli in 34 per cent of the stools of 78 individuals in Peking. May it not be assumed that here we have an instance of a reaction being set up by spores absorbed during digestion and carried to the liver? Obviously, the nature of the bacillus, the character of the food the species of animal, may all cause wide variation. The fatty and waxy coating of the tubercle bacillus, for example, makes it particularly resistant to the intracellular ferments of the leukocytes.

The question as to the reaction set up in the liver by the lodged bacteria is equally interesting. That bacteria, without the signs of tissue reaction, can be found in both the liver and the gall-bladder has been pointed out by Aschoff.[21] He had a large number of unchosen livers examined for possible inflammatory reactions. He concludes that although staphylococci, streptococci and pneumococci can be more or less frequently demonstrated bacteriologically, yet the gall-bladder and the intrahepatic bile passages may be normal in appearance and, therefore, that certain bacteria may be excreted through the liver and perhaps through the gall-bladder without a special reaction.

Adami[22] pointed out that, in conditions of congestion and slight chronic inflammation, bacteria may, time after time, pass through the intestinal wall to be deposited and destroyed in the liver. The microbes may not necessarily multiply in the organ. Repeated inroads and repeated destruction of bacteria, however, yield enough toxic substances to produce an appreciable alteration in the parenchymatous cells. He suggested the term subinfection to describe the reaction.

The reaction in the reticulo-endothelial cells, when considerable numbers of microbes are introduced into the blood, has been made the subject of an interesting study by Oerskov.[23] He used more or less virulent strains of streptococcus, staphylococcus, pneumococcus and Bacillus coli. He destroyed the animals at varying intervals from five minutes to several days after the injection. He studied the amount of phagocytosis by the Kupffer cells and the amount of exudation of leukocytes. He found in animals killed after two minutes many cocci in the liver and a few in the spleen. The cocci

were largely inside the Kupffer cells. The leukocytes were few. In an hour and a half all the Kupffer cells were packed with cocci and there was a considerable exudation of leukocytes. At the end of six hours the liver contained a great number of cocci and the leukocytes were in great numbers. At the end of eighteen hours there were still a large number of microbes in the liver but many of them stained poorly; the leukocytes were numerous. At the end of eight days the microbes were no longer found. In the destruction of the cocci by the Kupffer cells there was pronounced infiltration of the interstitial tissue with leukocytes; similar results were obtained in studying the destruction of other microbes. None of the polymorphonuclear leukocytes showed evidence of phagocytic activity.

In another series he introduced a fatal dose of pneumococci. The results were similar up to a certain time. The cocci were rapidly arrested and destroyed in the liver by the Kupffer cells, accompanied by the same infiltration of the interstitial tissue by leukocytes, but at the end of twenty-four hours there was a multiplication of the pneumococci. The protective cells were unable to hold in check the microparasites and the body was invaded.

According to the type of organism and the degree of immunity established in the body, not only an exudative inflammation occurs but various forms of degeneration, necrosis and regeneration, and various forms of productive inflammation, giving the most varied picture of interstitial and parenchymatous lesions.

Chronic productive inflammation of the interstitial tissue is very frequent and leads to a variety of alterations in the liver tissue which are grouped under the general heading cirrhosis.

There is a wide margin of safety in the liver. Rous and McMaster[24] have shown, in the dog and the monkey, that three-fourths of the secreting cells of the liver can be shut off without producing symptoms. That considerable cirrhosis can exist without clinical symptoms has long been well recognized.[25]

There can be no question that the liver is concerned in the defense mechanism of the body. It is concerned normally in the disposal of and making innocuous various substances brought to it; in the exaggeration of this function various reactions occur which are shown morphologically. The liver seems to be one of the first organs to react and form a first line of defense, as it were, in the protection of the body against disseminated bacteria.

Many of these reactions pass without evidence of disturbance,

such as fever, etc., which can be recognized clinically. There is an interesting hypothesis of Vaughan[26] in this connection. He suggests that when bacteria are destroyed, the general body reaction, the heightened metabolism, the rise of body temperature, all the general disturbances that we group under fever, are due to the disintegration of foreign bacterial proteins by extracellular ferments. The destruction of bacteria inside the cells may be unaccompanied by these phenomena. The intracellular ferments in the macrophages (von Kupffer cells) carry perhaps the destruction of the bacterial proteins to a point where the products are much less toxic or build them up into their own protein. The process of intracellular digestion may pass so smoothly that the host may be unaware of the bacterial destruction going on and present no disturbance of the normal working of the body that can be recognized clinically.

The large phagocytic cells of the reticulo-endothelial system have another function. They are concerned in the destruction and disposal of red blood cells. This has been shown to take place normally in the liver, the spleen, the bone-marrow and the lymphatic nodes. The hemoglobin is split into an iron-containing portion and and an iron-free portion, and the liver seems to play an important part in the disposal of both portions. When the red cells are destroyed in excess the iron accumulates in the liver. The iron produced in excess in the reticulo-endothelial system is released into the circulation and stores and slowly secreted by the liver cells. It shares this function of storage with the spleen and takes it over when the spleen is removed.

The disposal of the iron-free portion has been the subject of much study. It is concerned in the production of bilirubin. There has been much controversy over the site of the formation of bile pigment and the production of jaundice. In the well-known experiments of Naunyn and Minkowski[27] the liver was removed from a goose; then the bird was poisoned with arseniureted hydrogen. No icterus followed. In another experiment the liver was not removed. In this instance, poisoning by arseniureted hydrogen resulted in an enormous phagocytosis of erythrocytes and the production of jaundice. The text-books of physiology of fifteen years ago record the famous experiments. But as almost all the reticulo-endothelial cells in birds are in the liver, the spleen and bone-marrow containing relatively few cells of this system, these experiments were inconclusive when applied to mammalia.

Later, Whipple and Hooper[28,29] excluded the portal circulation in dogs, injected hemoglobin and were then able to show bile pigment in the urine, and Mann and Magath[30] observed the formation of bilirubin in dogs after the removal of the liver. The text-books of physiology of the future will record these experiments. It seems to be established today that one of the functions of the reticulo-endothelial system is the production of bilirubin. The Kupffer cells of the liver make up an important part of this system, so that although there is an extrahepatic formation of bilirubin, it by no means implies that the liver is not the seat of a considerable portion of bile-pigment production. Of the polyhedral cells of the liver it may perhaps be said that the evidence today is against their ever sharing in the formation of bilirubin. They excrete the pigment but do not form it. The bilirubin made by the reticulo-endothelial cells seems to be slightly different from the bilirubin secreted by the liver. van den Bergh[31] showed that these differences could be detected by the behavior of the pigment in the diazo reaction. These differences are possibly due to physical changes in the state of the bilirubin in the plasma. The bilirubin acted on by the liver cells diffuses more readily and is more readily oxidized. It gives what is known as the immediate or direct reaction. The bilirubin as it is formed by the cells of the reticulo-endothelial system diffuses less readily and is less readily oxidized. It gives a delayed or indirect reaction.

Concerning the secretion of bile salts and cholesterin, little definite is known. It is assumed by Brulé[32] and a number of French observers that the polyhedral cells have a selective action, very much as have the secreting cells of the kidney. At times, when the cells are damaged, now one, now another ingredient is not excreted by the polyhedral cells, so that there may be the presence of bile salts alone or bilirubin alone in the blood. There is not always a complete retention of all the component parts of the bile. Unfortunately the tests for the detection of the bile salts in the urine are open to criticism. However, his work is interesting and suggestive. The itching, the brachycardia, the absorption of fat, are all said to be due to bile salts. He thinks obstructive jaundice is "globale et brutale;" both salts are retained completely and suddenly. In hepatitis from various toxic substances the dissociation of the two substances found in the bile exists, now one, now the other appearing in the serum, then both appear and again in the disappearance of the jaundice there is a similar irregularity.

The liver is, as we have said, concerned in the storage and detoxication of various substances besides bacteria. When colloidal metals are injected into the circulation they are taken up by the Kupffer cells. If the dosage is sufficient the cells are injured. The damaged function is made evident by the clinical sign of jaundice, and the extent and character of the jaundice is, in a way, a measure of the impairment of the liver cells. Cunningham,[33] in studying the clinical effects of colloidal lead in patients to whom it had been administered for inoperable carcinomata, describes three forms of jaundice:

1. There is a slight icteric change in the sclera with increased urobilin and a positive indirect or delayed direct van den Bergh reaction. The bile-secreting cells are probably unaffected. The jaundice is due to excessive production of hemolysis by the von Kupffer cells and the reticulo-endothelial system. The pigment is produced in two large quantities to be taken up directly and secreted by the polyhedral cells of the liver parenchyma, with the result that a quantity continues to circulate in the blood.

2. There is damage to the polyhedral cells of the liver, urobilin and bile appear in the urine, the jaundice is deeper, the van den Bergh reaction is biphasic or of direct type.

3. In the third type there is evidently a cholangitis in addition, due to the destruction of the polygonal cells. Jaundice is deeper and there are large amounts of bile in the urine. There may or may not be an increase of bilirubin of the urine; the van den Bergh test is immediate or direct.

These studies seem to me of especial interest. They furnish an indication of the way certain toxic substances act in producing jaundice by direct action on the reticulo-endothelial cells and on the polyhedral cells of the liver. We have in jaundice an obvious clinical sign and, in certain cases, an indication of the disturbance of function, either in the secreting polygonal cells of the liver or in the Kupffer cells. A similar relation of jaundice to the irritation of microbes deposited in the liver exists.

In 1916 Inada and Ido[34] and their coworkers showed the presence of spirochetæ in the blood, liver and kidneys of patients suffering from an epidemic and an endemic form of infectious jaundice occurring in Japan. They transmitted the spirochetæ by intraperitoneal injections in guinea-pigs and cultivated the microparasites outside the body.

The action of the spirochetæ in producing the jaundice cannot be easily explained by obstruction of the bile passages. In the severe cases the bile passages are pervious. The lesions are confined to the hepatic cells. During the first days of the disease, before the appearance of the jaundice, the spirochetæ can be demonstrated in the blood. But the spirochetæ do not remain long in the general circulation. From the fifth to the eighth day, inoculations of guinea-pigs with the blood are negative; enough immunity is established to destroy the parasites in the circulating blood. The microparasites are found in the liver and other organs. Moreover, all grades of jaundice are produced. In certain cases there is no jaundice; in others it is slight and transitory, like a mild grade of catarrhal jaundice; in others it is severe and gives the picture of acute yellow atrophy. In reading the accounts of the disease it is difficult not to see a resemblance to the occasional cases of so-called acute catarrhal jaundice, and there has been a general tendency during the last ten years to place fewer and fewer cases under the heading of catarrhal jaundice. The majority are assumed to be sporadic cases of infective hepatitis. Not that there is any general assumption of a spirochetal origin for all forms of epidemic jaundice. In an article by Blumer,[35] in 1923, he expressly states that in the types of the disease which he has had an opportunity to see, no spirochetæ were demonstrated, and that he believed that they were not the causative agents in the types usually seen in this country. But the relation of epidemic forms of jaundice to catarrhal jaundice is too striking not to assume that the ordinary forms of catarrhal jaundice are but sporadic cases of infectious jaundice, and the resemblance of the epidemic infectious jaundice is too similar to the disease produced by spirochetæ not to be noted. The clinical picture of the fatal cases, which are rare, is that of acute yellow atrophy, just as in spirochetal jaundice, and, here again, all the evidence seems to point to hepatitis as the cause of jaundice. It is understood, of course, that acute yellow atrophy is not a disease entity but a syndrome produced by a number of toxic agents.

The autopsies which showed any lesions which corresponded to older theories of catarrhal jaundice are very few. The case reported by Eppinger[36] is of interest. A girl, nineteen years of age, suffering from acute catarrhal jaundice, in a fit of despondency threw herself out of a window. At autopsy the inferior extremity of the common duct was found blocked by a mass of inflamed lymphoid tissue.

The evidence today makes it probable that a large group of transitory jaundices are due to damage to the liver cells, not to obstruction from catarrhal inflammation. Many microbes set up an infective hepatitis and certain of them have an elective affinity for the liver, localize there and create lesions more or less grave.

In most of the acute infections there is a period when the microparasites are found in the blood stream. Here again, after a period during which a certain degree of immunity has been established, they disappear and are found deposited in the liver and other organs. In typhoid, in pneumonia, in secondary syphilis, in malaria, etc., jaundice is occasionally present. In streptococcal sepsis and other forms of sepsis, jaundice occasionally occurs. In the fatal cases at autopsy, aside from various parenchymatous and degenerative changes, the liver shows frequently a round-celled infiltration in the interstitial tissue and focal necrosis.

Here, again, it is difficult not to believe that some degree of hepatitis is almost always present and that, now and again, either due to some special weakness, predisposition or sensitization, the parenchyma cells are enough involved to interfere with their normal function of excreting bile. The obvious clinical sign of jaundice occurring every now and again in the course of disseminated bacterial invasion, is an indication of the hepatitis which is, in all probability, frequently present and is comparable to the jaundice seen occasionally during the administration of certain toxic substances.

It is interesting to see how often transitory infective icterus appears in subjects in which the liver is already the seat of a lesion, from alcohol, syphilis or pregnancy. Possibly toxic substances set free in the violent reaction of the Kupffer cells to the multiplying microbes produce changes in the polygonal cells, disturb their function and hinder secretion. The margin of safety is so great in the liver that such hepatitis must be widespread to produce jaundice. The study of the pathogenesis of spirochetosis has transformed the significance of jaundice. It has placed a large group formerly classed as obstructive jaundice under the jaundices due to action of microörganisms on the liver cells, and has called attention to the reaction of the liver cells regularly present in disseminated infection. Even in obstructive jaundice, where the main excretory ducts are closed, the liver cells are rapidly implicated. If there is obstruction of the hepatic duct above the point where the gall-bladder enters or if the common duct is completely shut off, after the gall-bladder

has been removed, there may be such a disturbance of the liver cells that no bilirubin is secreted. The ducts contain whitish matter made up only of the excretions of their walls, but the failure of the liver cells to secrete bilirubin is not due to failure of the pigment to be formed. The evidence all goes to show that the bile suppression is due to a disturbance of the liver cells, and only indirectly to an obstruction to the outflow. If the gall-bladder is in place, then the back pressure on the secreting cells is not so great and the damage is much less.[37] If the obstruction is intermittent, infection, either ascending or descending, inevitably follows and widespread cholangitis with further involvement of the liver cells occurs.

Various microbes not only settle out and are taken up by the liver cells in the course of disseminated infection but reach the interstitial tissue of the liver in clumps or masses from a disintegrating, infected clot in the portal vein or by emboli through the hepatic artery, being arrested in the small vessels and producing a variety of focal lesions and multiple or single abscesses.

It has long been recognized that patients suffering from amebic dysentery not infrequently develop liver abscesses. The amebae spread in the submucosa of the intestines. They may be found both in the bloodvessels and lymphatics, to be carried to the mesenteric lymph glands or through the portal circulation to the liver, where they set up a hepatitis. The hepatitis may subside or, if the tissue resistance is poor or the dosage massive, abscesses form. Here again the liver is the first great barrier to the dissemination of the infecting agent.[38]

In many of the stages of infection when a partial immunity has been established, various forms of hepatitis occur, often giving little or no clinical evidence of their presence. According to Calmette,[39] during the course of infection with tubercle bacilli, small tuberculous lesions are nearly always present in the liver if careful enough search is made. In an autopsy reported by Sabrazes,[40] in a patient who died of leprosy, the liver showed no leprous nodules but sections showed an enormous phagocytic reaction of the Kupffer cells. There were no bacilli, however, in the biliary passages; none in the bile. In late stages of syphilis the liver more frequently harbors treponemata than any other organ. Various forms of interstitial hepatitis, as well as the characteristic gummata, are present. Syphilis is relatively frequent.

In over 9000 Wassermann tests taken as a routine measure in all patients treated at St. Luke's Hospital during the last year, including the O. P. D., over 5 per cent showed a positive reaction. As in a number of these repeated examinations were made, and as there must be a large number with syphilitic infection, yet showing a negative examination, the actual percentage should be put much higher. It would be interesting if we knew in how many of these patients there were changes in the liver due to the lodged treponemata.

Anyone connected with a large hospital has an opportunity of seeing all these forms of hepatitis. He gets an impression of an extraordinarily complex etiology.

With these facts in mind, I shall next attempt to study the path of infection suggested in a number of recent papers and the relation of hepatitis to cholecystitis and then of cholecystitis to hepatitis.

In the first place there is a widespread notion that appendicitis, hepatitis and cholecystitis are related, and that infection passes from the appendix to the liver through the portal vein. That this relation occasionally exists, in acute cases in which there is a suppurative lesion of the veins in the mesentery of the appendix and a progressive suppurative pylephlebitis is set up, which leads to infected clots lodging in the liver, is generally recognized. But during the course of acute appendicitis, cholecystitis and hepatitis are not usually recognized. The associated acute lesions occur, but they are not common. I do not find, in patients operated on for acute suppurative appendicitis, any disproportionate number later developing clinically recognizable forms of hepatitis; nor do the autopsy records on patients dying of acute appendicitis as a rule show lesions of the liver. Tens of thousands of microörganisms are undoubtedly lodged in the liver, just as they are in the spleen and the bone-marrow in general sepsis from acute appendicitis. They are taken out of circulation and many of them are destroyed in these situations. Nor is the proof offered that hepatitis has been found, in removing sections of liver while performing appendectomy for chronic appendicitis, convincing. The sources of error are obviously too numerous; the impossibility of excluding other inciting agents is too great; the number of observations are too few.

Nor do I find evidence that cholecystitis regularly follows hepatitis. In the numerous instances of infective hepatitis I can find only occasional instances of involvement of the gall-bladder which

reaches the grade of clinical recognition. Although hundreds of thousands of microbes are lodged in the liver there is only very exceptional evidence of acute cholecystitis. I do not question that if these patients were operated on, various slight degrees of round-celled infiltration of the wall of the gall-bladder might be found, but during the course of the jaundice that is so frequently present in infective hepatitis there is rarely present a form of cholecystitis that can be recognized clinically.

During the last year I have twice operated, through a mistake in diagnosis, on patients with infective hepatitis. A woman, aged thirty-seven years, entered the hospital acutely ill with a temperature and jaundice. She was kept under observation. There was a history of syphilitic infection and treatment by salvarsan five months previously, and, although she had had little pain, having only a sense of fulness and distention after meals, and gave no history of having had attacks suggesting cholecystitis, the erroneous conclusion was reached that she had an obstructive jaundice. On opening the abdomen the gall-bladder was full but could readily be emptied. There were no adhesions, the wall was thin, there was no dilatation of the ducts, the liver seemed a little firm. A section removed for examination showed the lesion of severe, extensive hepatitis, the lesion extending about the minute bile ducts and about the portal venules. The gall-bladder was anastomosed to the stomach. The patient made a good recovery; the jaundice very slowly subsided, as is the rule in infective jaundice. The patient, seen one year and a half later, was in good health, with no jaundice. There was marked hepatitis, proved by the microscopic section, and no appreciable cholecystitis. The second case was similar. There was hepatitis present; the gall-bladder and ducts seemed normal. The patient had an infective hepatitis imposed upon a cirrhosis of the liver.

The following case report is also interesting: A man, aged forty-one years, entered the hospital very ill, with fever and general abdominal pain. He was slightly jaundiced. The abdomen was opened through a right rectus incision; the gall-bladder seemed normal; a gangrenous retrocecal appendix was discovered and removed. He grew gradually more jaundiced and died on the second day. During the last day he complained bitterly of pain in his right leg. At autopsy the liver showed marked hepatitis. The common duct was patent and there was no dilatation. The

cystic duct was patent; there was no gross lesion of the gall-bladder. There was infection of the retroperitoneal tissue; the psoas muscle was soft and pultaceous. Cultures from the retroperitoneal infection showed a large nongas-forming bacillus. This patient is an example of hepatitis following appendicitis, yet advanced acute hepatitis was unaccompanied by gross lesions of the gall-bladder.

In typhoid fever the relation of hepatitis and cholecystitis has attracted much attention. The initial period of typhoid fever is accompanied by the presence of typhoid bacilli in the blood stream. After a time the bacilli are no longer found in the blood. They are found in the bone-marrow, spleen, lymphoid tissue of the intestine and lymph glands of the mesentery. In the lymphoid tissue of the intestine they colonize, multiply and produce the characteristic lesions of the disease. The livers of patients dying of typhoid regularly show changes: there is fatty degeneration, infiltration with leukocytes, masses of leukocytes about degenerated and fragmented liver cells. These nodules are said to be due to bacterial emboli.[41] At times there is acute diffuse hepatitis or massive fatty degeneration; some of these patients are jaundiced, and the jaundice, to quote Widal, is due to a hepatitis: "The jaundice of typhoid is really a hepatitis, the result of lesions of the hepatic cells, determined by the localization in the liver of microbes deposited there from the general circulation."[42] The bile regularly contains typhoid bacilli and there are all grades of lesions of the gall-bladder, from catarrhal to purulent inflammation, yet cholecystitis giving clinical symptoms is not common in typhoid fever. From the seventh to the thirtieth day, pain in the right hypochondrium, tenderness and rigidity are the symptoms that all who have an opportunity to see any considerable number of patients suffering with typhoid associate with the condition. Though thousands of microörganisms are in the blood stream and the liver, and although the gall-bladder wall frequently shows lesions, only occasionally do symptoms appear that bring the lesion into the plane of clinical observation. Other factors seem to be necessary. One would expect that occasionally bacterial emboli might be lodged in the wall of the gall-bladder. One would expect that occasionally bacteria would be carried by the lymph stream to the wall, but it is difficult not to believe that the thousands of organisms in the infected bile must play a most important part in producing lesions of the gall-bladder. The experiments of Nichols are suggestive.[43] He succeeded in 63 per cent in producing gall-

bladder lesions when injecting typhoid bacilli by the mesenteric vein and in 11 per cent when injecting into the systemic veins. There was a considerably higher percentage of gall-bladder infections occurring in the immune animals than in the normal animals. He brought forward the view that gall-bladder infections in inoculated animals are not necessarily an index of immunity, but may be in part an indication of a rich amount of immune bodies in the blood.

The colon bacillus, aside from infection of the liver following stones lodged in the common duct and consequent infective cholangitis, can, in exactly similar manner, cause jaundice. The following autopsy report is an example. The skin was jaundiced, the liver showed acute hepatitis, the common duct was normal in appearance, the gall-bladder showed a subacute lesion. There was extensive degeneration of the liver cells and central necrosis, yet the gall-bladder showed only a subacute lesion. Cultures taken from the gall-bladder bile showed colon bacilli.

In the purulent form of interstitial hepatitis it is curious to see the absence of correspondence in the lesions of a gall-bladder and the lesions of the liver. A child, ten years of age, very ill, was admitted to the hospital. She had a high temperature, tenderness and an indistinct feeling of mass in the right hypochondrium. The abdomen was opened, the liver was found enlarged, the gall-bladder seemed acutely inflamed. It was removed. The patient died in three days. At autopsy there was only a small amount of serous fluid in the upper part of the abdomen. There were no signs of peritonitis. The liver was enlarged and riddled with abscesses. There was an early parietal clot forming in the portal veins. Section of the gall-bladder removed at operation showed a subacute cholecystitis. Although the liver was filled with abscesses, some of them but a few centimeters from the gall-bladder, only enough bacteria had passed through the lymphatics to set up a subacute lesion. Cultures from the pus in the abscesses showed colon bacilli.

In amebic abscesses I have been unable to find records of gall-bladder involvement. Although tubercular lesions of the liver are said to be common, tuberculosis of the gall-bladder wall is rare. Simmonds[41] has given a description of the forms. In syphilis, although various forms of hepatitis are common, there is no evidence of unusual frequency of clinical forms of cholecystitis. As I understand the evidence, there is no unusual incident of gall-bladder infection following the various forms of hepatitis. Bacteria are

probably not infrequently present and not infrequently there are slight evidences of an inflammatory reaction. To produce a clinical form of cholecystitis, added factors are necessary.

If the gall-bladder is grossly infected, if the bacteria are lodged in its wall and the tissues react, and the lumen is filled with exudate and bile, does a form of hepatitis develop leading to clinical forms of cirrhosis? A reacting zone of contiguous structures is formed about the inflamed organ. The peritoneum, the wall of the colon and the duodenum are glued to the gall-bladder by exudate. The omentum turns up and is adherent to the gall-bladder and the edge of the liver; the cellular tissue between the gall-bladder and the liver reacts and the interstitial tissue of the liver reacts. The extent of the zone of circumscribing reaction depends, as in all infections, on the virulence of the lodged organisms and the local and general resistance. There are all grades of severity, from inflammatory changes that can hardly be detected by microscopic study, to changes where purulent exudate and ulceration and necrotic processes are the characteristic features; and in all of them the liver in contact with the gall-bladder shows varying grades of hepatitis. Moreover if the attacks are repeated there may be evidences of chronic interstitial inflammation. Sections taken of the quadrate lobe or the right lobe in the neighborhood of the infected gall-bladder regularly show signs of inflammation on microscopic examination.

Anyone who has operated on a number of gall-bladders recognizes, as a matter of course, the infiltration of the cellular tissue between the gall-bladder and the liver, succulent in fresh infection, dense and firm in long-standing cases. When the gall-bladder ulcerates, or is necrotic at some point, an abscess may form partially in the liver substance. There can be no question that some form of local hepatitis occurs in almost all infections of the gall-bladder and that the infection travels from the gall-bladder to the liver. It is a matter so generally accepted that it has rarely been emphasized. It is part of the pericholecystitis nearly always present. Specimens, however, have been taken in many instances far enough away from from the bed of the gall-bladder to indicate that the reaction is widespread. There is no means of knowing in these instances whether the reaction in the liver may not be due to one of the many irritants which reach the liver. This may readily be the case if the gall-bladder lesion does not present gross morphologic changes. In any event, the hepatitis which we see so

frequently at operations for cholecystitis does not seem to have clinical significance, for if the grossly inflamed gall-bladder is removed, there is no progressive involvement of the liver. In several cases in which I have removed a gall-bladder with an abscess between the gall-bladder and the liver, the infection in the liver has apparently promptly subsided, for these patients are in good health and fat when seen years after their operations.

A patient entered the hospital two years ago presenting several interesting features: He had a bottle in his hand containing gall-stones that he had passed twenty years ago. He had had, after this long period of latency, a severe attack of typical gall-bladder pain. Twenty-seven years ago he had had his first attack, and during seven years following had had a number, several of them with jaundice. During the last one of these attacks he passed the stones he showed. He was operated on by Dr. Bolling and an obviously inflamed gall-bladder was removed, containing stones in every way similar to the ones he had passed many years before. The liver seemed firm and the edge a little rounded. The wall of the gall-bladder showed a small carcinoma. He had at that time and still has, two years later, no signs of chronic hepatitis or liver cirrhosis that can be appreciated clinically, although there had been enough irritation to produce a new-growth, and although the irritating agent had acted over a period of twenty-seven years.

There is another bit of evidence worth recording. Fifteen years ago, in a number of the large clinics, the gall-bladders were not removed. The stones were removed and the gall-bladder drained. The practice was abandoned. There were too many recurrences due to impacted stones or stenosis of the cystic duct. Many of these patients recovered. I can recall no instance or report of the retained, grossly infected gall-bladder setting up chronic forms of hepatitis. I gained the impression at that time that a draining gall-bladder, even if infected, gave little or no symptoms.

In deeply jaundiced patients with septic fever and a stone in the common duct, I have in several instances left the shrunken gall-bladder in place, opened the common duct and removed the stones. In no instance has a progressive hepatitis or liver cirrhosis followed.

I do not find evidence, then, of a so-called vicious circle; a chronic appendicitis, then a hepatitis, then a cholecystitis, and the cholecystitis then becoming the main focus and producing hepatitis.

The evidence seems to be that the liver is a most efficient filter for bacteria, taking them up from the general or portal circulation, and that only a few pass the cells of the peculiar liver capillaries to reach the lymph and float along with the lymph current into the nodes at the hilus. At autopsy or at operation these nodes are not infrequently enlarged, but the enlargement of the lymphatic nodes is an inconspicuous feature of infective hepatitis. I do not question that a few bacteria may pass along the lymphatics from the liver into the wall of the gall-bladder. I do question, however, whether slight infiltration of the gall-bladder wall, changes so slight that it is impossible to recognize them by gross examination, are clinical forms of cholecystitis. This assumption seems to be based on the observation that certain patients gain relief from symptoms of a form of dyspepsia after the removal of a gall-bladder which shows no macroscopic lesions or shows beneath the mucosa, when cut open, small yellow spots from lipoid deposits (strawberry gall-bladder). This form of indigestion is shown by accumulations of gas in the upper abdomen, belching, sour regurgitations occurring promptly after eating a hearty meal or badly prepared or indigestible food.* Unfortunately these symptoms are present to some degree in most middle-aged people. They are present, as I have assured myself, at times after the gall-bladder has been removed and they are absent in many instances in which there is a gross lesion of the gall-bladder wall.

In the same way, study of the cholecystograms made by the method recently introduced by Graham has been said to furnish a surer means of recognizing gall-bladder disease than the sight and touch of the surgeon at operation; that is, by the observation of the filling and emptying of the gall-bladder one can determine whether a gall-bladder is so diseased that it should be removed, even if there is no demonstrable lesion at operation. The gall-bladder disease is proved by the microscopic examination of the excised gall-bladder wall showing a very slight cellular infiltration and by statements made by the patients of relief after operation. But is it not too early to draw this conclusion; is not too little known of the normal function to speak so confidently of abnormal functions? I grant that certain patients get complete relief after operations for the removal of the gall-bladder with very trifling lesions of the gall-bladder wall, but a more or less lasting freedom from symptoms has been presented as proof for all manner of remedies. Patients

treated by homeopathy and osteopathy are sincere in their belief in the remedies applied and grateful for the relief afforded. I believe we should demand stronger proof. I have an instinctive distrust of occult surgery or surgery for occult lesions. I recently saw a middle-aged woman operated on in a large clinic. Her symptoms were, as nearly as I can remember, gas, indefinite gastric discomfort, inability to eat certain foods, and pain after eating. When the gall-bladder was exposed it was soft, thin-walled, on manipulation it emptied, there were no adhesions, no stones. The gall-bladder takes up and concentrates bile and regulates the pressure in the biliary system.[17] If the orifice of the cystic duct is blocked, there is evidence of disturbance and pain. The gall-bladder in question emptied, it was thin-walled and distensible. It contained stasis bile. Which of its known functions was at fault? The gall-bladder was removed, it was cut open, and from the appearance of the thick, stringy stasis bile, cholecystitis was said to be present.

The pathologist, Aschoff,[18] whose painstaking and thorough studies have thrown so much light on the formation of gall-stones and the function and structure of the extrahepatic bile passages, has pointed out that in studying postmortem staining of the biliary passages there are fairly constant findings. The region of the sphincter of Oddi is usually pale, almost colorless, a sign that it is closed, even after death. The common and hepatic ducts and the cystic duct up to the neck of the gall-bladder are stained yellow, that is, stained with hepatic bile, while the neck and the rest of the gall-bladder are stained dark brown by the stasis bile of the gall-bladder, as if the change in color marked a division between the conducting system and the condensing system of the extrahepatic bile passages. He also points out that there is sphincter-like thickening of the smooth muscles at the beginning of the cystic duct and numerous ganglion cells and nerve fibers in the wall of the cystic duct. We apparently have to think not only of the mechanism of control at the orifice of the common duct but of a mechanism of control at the outlet of the gall-bladder. That among the patients in whom the gall-bladder has been removed for trifling lesions and in whom the cysticus is patent there are a few who have had definite cramp-like attacks due to disturbance of the nervous control mechanism which synchronizes the various sphincter-like structures at the orifice of the cystic and at the orifice of the common duct,

seems very probable. But in the great majority of cases, to produce a clinical form of cholecystitis, either very unusual virulence of the microörganisms is necessary or there is added to the factor of slight infection a disturbance either mechanical (calculus, anatomic peculiarities, etc.) or functional, which interferes with the passage of the gall-bladder contents through the cystic duct.

SUMMARY. 1. One of the main functions of the liver is the destruction and disposal of bacteria and toxic substances.

2. Bacteria and toxic material reach the liver in a great number of ways and very frequently throughout life.

3. Some are destroyed there without appreciable reaction and all grades of appreciable reaction occur.

4. The morphologic changes which occur in the liver in the reaction of the cells to irritants, and which are called by the pathologist hepatitis, are extremely difficult to classify. The subject of hepatitis is one of the most complex in the domain of pathology. It is in no sense an entity, nor can it be discussed with profit as an entity.

5. Many of the cases formerly grouped under acute catarrhal jaundice are now considered forms of infectious hepatitis.

6. In the forms of hepatitis which have reached the level of clinical observation there is little evidence of a relation to clinical forms of cholecystitis.

7. The hepatitis regularly found with cholecystitis has little or no clinical significance and is not a factor of importance in causing clinical forms of liver cirrhosis.

8. The proof that very slight infiltration of the gall-bladder wall and lipoid deposit in the mucosa cause acid indigestion, a feeling of fulness in the epigastrium, flatulence, intermittent gastric pain after eating badly prepared food, is by no means conclusive.

In closing, may I quote the following letter of advice to a young noblewoman from Burton's *Anatomy of Melancholy:* "In this hypochondriacal or flatuous melancholy the symptoms are so ambiguous that the most exquisite physicians cannot determine of the part affected." The symptoms of this disease are said to be "sharp belchings, fulsome crudities, heat in the bowels, wind and rumbling in the guts, vehement gripings, pain in the belly and stomach sometimes, after meat that is hard of concoction." I am not sure

whether Burton's rendering is better than the original Latin, which I add: "Acidi ructus, eruditates, æstus in præcordiis, flatus, interdum ventriculi dolores vehementes, sumptoque cibo concoctu difficili."

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APPENDIX

INTRODUCTORY NOTE TO THE SECTIONS

Almost all of the papers read before the various sections of the College are subsequently published in full in medical periodicals.

The papers listed in the following pages may be found by consulting the *Index Medicus*, provided a sufficient length of time has elapsed from the date of publication to the next succeeding quarterly number of the *Index Medicus*.—[Editor.]

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY, 1926, TO DECEMBER, 1926

JANUARY 21

Bilateral Paralysis of the Third and Fourth Nerves

Dr. Leighton F. Appleman exhibited a case of bilateral paralysis of the third and fourth nerves which came under observation during the previous week. When first seen, the paralysis was more complete in the left eye, although within five days after he was first seen the right eye was equally affected, with ptosis, moderate dilatation of the pupils, which were unresponsive to light, and loss of accommodation and convergence. The patient gave a history of specific infection for which he had previously received treatment. The Wassermann reaction was still positive.

He was placed upon nonspecific therapy, receiving one injection of Coley's fluid up to the present time, as the result of which there is a distinct lessening of the ptosis upon the left side.

The patient is of interest clinically, and at a future meeting may again be shown in order that the results of this form of therapy may be evaluated and a further and more detailed study made available.

A Case of Extensive Nævus of the Eyeball

Dr. William Zentmayer exhibited the drawings of a case of extensive nævus of the eyeball in a colored woman, aged fifty-two years. The case was shown before the Section at the October meeting in 1922.

At that time the pigmented area began at a point about 4 o'clock and encircled the cornea to about 8 o'clock. Except for its color, which was a chocolate brown, it resembled very much a subconjunctival hemorrhage, having the same distribution and arrangement. At two locations it had invaded the cornea. The patient stated that there had been a spot on the eye from birth, but that it had recently been increasing and causing discomfort.

It was thought that it might be developing malignancy and that desiccation or some similar procedure had better be done. Dr. William L. Clark, to whom the patient was sent, hesitated to do this at the time and thought it would be better to wait and see whether it continued to grow.

There has been considerable spreading of the pigment since the above date, and at the present time it encircles the cornea except for a small break of 2 mm. between 12 and 1 o'clock. It has also advanced into the corneal tissue almost around its entire circumference. It seems also to have extended a little further in the palpebral conjunctive of the lower lid.

The eye has normal vision and normal fields. The refraction is O.D. -0.25 sph. $= +0.75$ cyl. ax. 150 degrees; O.S. $+0.25$ sph. $= -0.75$ cyl. ax. 105 degrees.

Aside from the unusual extent of this nævus, if such it be, the case is of interest because of its taking on growth after long years of quiescence and extending into the cornea.

The nature of the growth is difficult to determine without microscopic examination. Probably the term melanosis of the conjunctiva would better describe it.

Sudden Unilateral Blindness following Injury

Dr. Warren S. Reese reported this case with the thought that it might be of interest from the standpoint of diagnosis and also in connection with Dr. Adler's paper upon pupillary reactions.

This man was riding on a motor car on the Reading Railroad on December 19, 1925, when the front wheels flew off. He was thrown on the track, striking his head in the right frontotemporal region. He was not rendered unconscious, but, on arising, noticed that he could not see with the right eye. When seen two days later he was totally blind in the right eye. There was a sutured laceration through the right brow and a smaller one of the lower lid. There

was no proptosis, chemosis or subconjunctival hemorrhage. The direct-light pupillary reflex was absent, but the consensual reflex was normal. An *x*-ray for fracture of the orbit was negative.

Dr. Reese considered the most likely cause of this blindness to be fracture involving the right orbit. Another possibility was hemorrhage into the optic nerve sheath. Absence of signs of fracture and a negative *x*-ray made the latter seem more likely. The pupil disturbance ruled out malingering. Another cause that recently occurred to him was the extension of a subperiosteal hemorrhage back to the optic foramen, thus causing a compression of the optic nerve. Dr. J. Parsons Schaeffer said that this was very possible. He also suggested hemorrhage into the sphenoid sinus as a possibility. The patient now has an advanced atrophy. There has been no change in the vision or pupillary reaction.

Discussion. Dr. William Zentmayer asked, "What was the condition of the retinal arteries when the patient was first seen?" If they were attenuated it was suggestive of a hemorrhage within the sheath of the nerve. That a fracture at the apex can be produced by a blow on the orbit was shown experimentally some years ago by an English ophthalmologist.

Dr. Luther C. Peter believed it was possible to rule out subperiosteal hemorrhage in Dr. Reese's interesting case as an etiological factor. As there were no motor symptoms, it is not likely that subperiosteal hemorrhage would produce optic atrophy. On the other hand, it is rather interesting to note the extensive motor involvement which may follow subperiosteal hemorrhage in the orbit without the presence of an atrophy. He recalled a case of his in which there was complete internal and external ophthalmoplegia following a blow on the eye from an exploding tire. This patient made a complete recovery with the exception of slight paralysis of the external rectus. There was no optic atrophy at any time, vision and fields remaining normal. He was inclined to think that the blindness and subsequent atrophy were due to damage of the nerve by "contra coup." The *x*-ray examinations of the optic foramen, as a rule, are not well done. von der Hoeve, several years ago, pointed out the necessity for a special technic. Roentgenologists are rather indifferent to the use of this technic, which is absolutely essential in order to fairly outline the optic foramen. Plates exposed in this way are more apt to show lines of fracture running off from the optic foramen than plates taken by the usual method. It is not unlikely that in Dr. Reese's case an examination made by this method might have shown a line of fracture.

Dr. Zentmayer stated that there are cases on record where this lesion produced a blindness, but he was unable to state whether or not, in connection with the amaurosis, there were ocular palsies.

Dr. William M. Sweet said that in a number of cases examined some years ago by the *x*-rays, he had never been able to demonstrate a fracture through

the orbital foramen. He had seen 2 cases of concussion injury in which blindness followed in both eyes; one, a man working at an iron-puddling furnace, the iron bar striking the side of the head, causing him to fall and strike the other side of the head on a pile of iron; and the other received a glancing blow on the front of the head from a mass of iron falling from a crane.

Dr. Reese, in answer to Dr. Zentmayer, said that there were no ophthalmoscopic signs to account for the blindness. There was some fuzziness of the nerve and distention of the retinal veins. According to Parsons, hemorrhage into the nerve sheath plays a large part in injury to the nerve, v. Holder finding it in 42 out of 54 cases of fracture.

Macular Edema with Paritial Separation of the Retina

Dr. Luther C. Peter presented report of a patient, a physician, sixty-six years of age, who, about the middle of last October, noticed a blur before the left eye, the blurred area being definitely outlined and greenish in color. Objects were distorted. Vision rapidly fell from 6/5 to 6/15 on October 20, 6/30 on October 26, and 6/45 on November 4. The patient described a definite, brownish, circular scotoma, which at first was of a greenish hue.

A careful physical and laboratory examination showed a negative Wassermann, normal blood chemistry, negative nasal sinuses, a normal urinalysis, blood-pressure of 135 over 85, and a cardiac condition which might be regarded as normal for a man of his age with a slight tendency to fibrosis. One tonsil showed a small pus pocket containing streptococci and staphylococci. There was a history of an old duodenal ulcer of many years ago. No trauma.

Examination of the fundus did not disclose any macular disturbance. When, however, the patient was examined with the simplified Gullstrand, there was found a definite elevation of the macular region. The elevation was rounded and symmetrical, and of the same color as the surrounding retina. The vessels were definitely observed to climb up over the domelike elevation. The stereoscopic view afforded by the Gullstrand ophthalmoscope left no doubt as to the pathology. The border of the elevation was slightly pale and a small patch of exudate was observed at the lower border. Red-free light did not give the characteristic picture of the macula either with the Gullstrand or the arc light, and red-free light did not give the characteristic picture of the macula. Following this examination, it was possible, by careful focusing, to observe a

difference of three diopters in the level of the small vessels at the top of the dome, and those of the surrounding retina.

At this time it was observed that with the addition of a +2.50 sphere the patient could read 6/7.5.

Although laboratory and clinical tests were largely negative, the patient was treated with hot packs, eliminative treatment and rest. Ten days later, vision improved slightly, and, by means of the Gullstrand, it was observed that the upper part of the dome was depressed, while the lower part was more bulging -the elevation being manifestly of a fluid character. This treatment was continued, with slow but steady improvement. At the last examination, December 29, 1925, a correction of +0.50 sph. = +0.50 c. ax. 190 degrees brought vision up to 6/6. At this last examination the macular area could be seen emerging from the upper part of the swelling.

Points of Interest. The case is of interest from the standpoint of diagnosis as well as from other features of less importance.

As to diagnosis, there manifestly is a partial separation of the retina from the choroid, and infiltration of the retina. The color and positive character of the scotoma, the distortion of objects and the elevation of the entire macula, as shown by the refraction, as well as the early bladder-like elevation are all characteristic of retinal separation. The green vision and the positive character of the scotoma are symptoms which definitely differentiate the condition from a possible choroidal separation. The preservation of macular vision, although somewhat lowered, must be explained by a nutritive supply which has not been completely cut off.

Green vision is a symptom to which our attention has been called as a forerunner of retinal detachment.

Of no less interest is the positive scotoma. In both editions of *The Principles and Practice of Perimetry*, Dr. Peter has stated that a positive scotoma may be produced by a cutting-off of the nutritive supply of the outer neuron of the retina, *i. e.*, the rods and cones, as well as by an interference with rays of light in reaching the rods and cones, as by hemorrhage or exudate anterior to this layer of the retina. This statement has been questioned by some. The case under discussion is a concrete example of the formation of a positive scotoma by this means. The macula is elevated from two and a half diopters, as shown by, first, the refractive measurement of the elevation by means of the ophthalmoscope, and sometime

later by measurements of macular vision by means of the trial case. From an etiological standpoint the case is also unusual. There is little to explain the cause of the condition. It is probable, however, that it is of cardiovascular origin. The gradual recovery and improvement observed under treatment would tend to confirm our conclusion in this respect.

Finally, it is interesting to note the value of the Gullstrand binocular ophthalmoscope in uncovering what the monocular ophthalmoscope fails to disclose. After the Gullstrand disclosed the elevation, one could, by very careful observation, determine the presence of an elevation. With the monocular vision, however, one is obliged to call upon the imagination, whereas the binocular instrument leaves nothing to the imagination to construct. A lesion stands out in relief or in depth.

Presentation of a Set of Couching Needles. Historical Sketch

Dr. Hunter H. McGuire, of Winchester, Virginia, presented to the Section a set of couching needles used by his grandfather, Dr. Hugh H. McGuire, in the performance of the cataract operation by the method in vogue during the early and middle portion of the Nineteenth Century. This gift was accompanied by a historical sketch of Dr. Hugh H. McGuire, of Winchester, Virginia, who attained a wide reputation as a surgeon, and especially as an eye surgeon, throughout northern Virginia.

FEBRUARY 18

Case of Bilateral Uveitis with Complicating Cataracts

Dr. Burton Chance and Dr. H. J. Blackmon presented a case of bilateral uveitis which began in November, 1924, with gradually failing vision in each eye and recurring attacks of pain and redness. For the past three months the patient has been unable to see well enough to get around.

Examination of the right eye shows an organized exudate, with considerable vascularization, in the lower anterior chamber. This exudate is about 4 mm. wide below. The iris is attached to the

exudate posteriorly. There are evidences of old iritis, with complete annular synechia. The lens is completely cataractous.

The left eye is similar except for numerous isolated, grayish deposits on the entire posterior surface of the cornea. Also, the organized exudate is less and the iris is less adherent.

The tension in O.D. is 8; in O.S. 11. Light projection is good. The blood-pressure, 150 SS. Nose and throat examination shows bilateral purulent ethmoiditis. Other findings are negative, including the Wassermann. However, he was under specific treatment for several months before admission. Cataract extraction is to be done on the left eye within a few days.

A Case of Paralysis of the Inferior Oblique Corrected by Tenotomy of the Superior Rectus of the Other Eye

Dr. William Zentmayer exhibited a case of isolated palsy of the inferior oblique muscle of the right eye, referred to him by Dr. C. P. Franklin, of the Veterans' Bureau, upon which he had performed tenotomy of the left superior rectus for the correction of the diplopia.

He pointed out that this was a procedure recommended years ago by Mauthner; but that Landholt, adhering to his preference for advancements in all ocular muscular anomalies, advises advancement of the inferior rectus muscle. As is stated by Duane, the inferior oblique of one eye and the superior rectus of the opposite eye are complete physiologic associates. The left superior rectus and the right inferior oblique have a common field of action, the left upper quadrant. In this common field, and elsewhere too, the inferior oblique moves the right eye up in precisely the same way and nearly to the same amount as the superior rectus moves the left eye. Each acts to move the eye up, each rotates the eye slightly to the right, each tilts the vertical meridian to the right, and the elevating power of each increases as the eyes are carried to the left. The lateral and torsion action of both muscles increases as the eyes are carried to the right and decreases as they are carried to the left.

In the present case there has been a slight overcorrection of the defect, as evidenced by the fact that the secondary deviation has been transferred from what was the sound eye to the affected eye.

There is, however, no spontaneous diplopia, but it can be elicited with a red glass, and the corneal images are symmetrical in all the fields of regard.

Discussion. Dr. Luther C. Peter stated that the operation which Dr. Zentmayer performed on this patient is the logical operation to correct the diplopia and relieve the patient's discomfort. Duane has demonstrated the value of operation on the associated muscle in paralysis of one of the vertical group. As Dr. Zentmayer has pointed out, the action of the superior rectus of the left eye is, in all respects, similar to that of the inferior oblique of the right. One would naturally expect, however, an overcorrection from tenotomy of the superior rectus muscle. While the inferior oblique has considerable power as an elevator, its elevating action is not equal to that of the superior rectus. This is due to the difference in the attachment of the superior rectus from that of the inferior oblique, forming an angle of about 23 degrees with the antroposterior axis of the eyeball in contrast to an angle of 39 degrees for the inferior oblique.

Dr. Warren S. Reese recalled that he had exhibited this patient here three years ago as a case of anophoria. From a rough examination at the present time, this condition apparently still obtains, although, as Dr. Zentmayer states, the left eye is not so markedly up under cover as before operation. There was formerly a marked rotatory movement of the right eye when uncovered. This has also been much benefited. Judging from his cursory examination, he believed that there is still a right hyperphoria when the Maddox rod is over the right eye, and a left hyperphoria when the Maddox rod is over the left eye.

Dr. Edward A. Shumway said that he had reported a case of traumatic paralysis of the left superior oblique which had been cured by tenotomy of the right inferior rectus muscle. Landolt's dictum had always been to advance the corresponding superior and inferior rectus muscle in these cases, but their position made advancement more difficult, and, as very good results were secured by tenotomy of the opposing muscle, this was usually the operation of choice.

Dr. Zentmayer, in closing, said that the operation of tenotomy of the inferior rectus for paralysis of the superior oblique of the opposite eye is probably more often performed. He recalled the first case he had seen so corrected, the operation having been performed by Dr. Norris. He himself had done the operation three times—twice for diplopia following a Killian operation, and once for a separation of the pulley of the superior oblique from a blow on the orbit, the diagnosis having been correctly made by a physician, Dr. Crabtree, who sent the patient to the hospital. He said that Dr. Norman Risley had also presented a case following a Killian operation. He could not quite understand Dr. Peter's statement that the inferior oblique has less elevating power than the superior rectus, because if this were so in their common field of action, the upper outer quadrant, there would be diplopia.

A Simplified Method of Complete Enucleation

Dr. Frederick Krauss showed a simplified method of complete enucleation, the basis of which was an accurate approximation of the ocular muscles and Tenon's capsule by silk sutures. The conjunctiva is stitched separately. In performing the operation, the conjunctiva is dissected in the usual manner. The tendon and attached extension of Tenon's capsule are caught upon a large strabismus hook, a needle threaded with No. 5 black silk is passed from above and under and to the lower third of the tendon, where it is passed through and is reintroduced into the upper third of the tendon, passing freely out under the lower edge of the tendon, making a loop in the tendon. The ends are caught with a hemostat, but not tied; the tendinous attachment is then cut as near the sclera as possible.

A similar procedure takes place in the opposite muscle; then the vertical muscles are caught in the same way. When all the muscles have been freed from the eyeball, saving as much of Tenon's capsule as can be obtained, the eyeball is removed in the usual way. As soon as fresh bleeding ceases, the tendons are approached from the opposite sides, by taking the first needle and thread through the opposite tendon, behind the location of the loop suture, and tying it to its mate. Similarly the needle of this second side is put through the tendon of the first side behind the loop and tied, making a firm union. The same is done with the vertical muscles.

When approximation is complete, the conjunctiva, which has been lying free, is united horizontally by interrupted sutures, making a clean socket. The result is very satisfactory. The buried sutures after a few weeks will come forth and are readily removed by simple traction. The movement of the eyeball and eyelids are remarkably preserved by this method.

Diabetics and Tobacco Amblyopia

Dr. G. E. de Schweinitz and Dr. A. G. Fewell, after some reference to the well-known vulnerability of the optic nerves of diabetics to tobacco, and after summarizing the case histories of several patients with diabetes who also were excessive smokers, and whose visual charts interpreted a chronic axial neuritis in which tobacco toxemia was evidently the prime factor in its development,

endeavored to show how these scotomas differed from those which were caused alone by the diabetes.

Their charts, which portrayed the "distinctive features" of the tobacco scotoma clearly, showed how it differed from that which was occasioned by diabetes alone, the patients being nonsmokers and abstainers from alcohol. They also pointed out that even moderate smoking in diabetics was harmful, as Leber long ago contended. They emphasized the importance of warning diabetics of this danger, and their cases indicated how failure on the part of the medical attendants had been responsible for an amblyopia which otherwise would have been avoided. Although a true diabetic amblyopia occurs, it is not a common condition, and in the majority of instances the axial degeneration is caused by tobacco or alcohol, the optic nerve being sensitized to tobacco by the diabetes. They also exhibited a series of field charts in cases of tobacco amblyopia unassociated with diabetes, and demonstrated how exactly they conformed with those of the diabetic patients who were smokers and who had acquired a so-called central amblyopia.

Discussion. Dr. Luther C. Peter observed that many points of interest might be discussed in this very excellent presentation of Dr. de Schweinitz. First, that of the predominance of the blue pericentral scotoma. There is a relation between the loss of blue and choroidal nutrition of the outer neuron of the retina, loss of red and green being especially identified with the transmitting part of the neuron or nerve fibers themselves. When there is a preponderance, therefore, of a blue scotoma, one would naturally think of nutritional disturbances rather than a pure toxic amblyopia. Second, the differences in results as to the size and shape, extent and saturation of the scotoma in toxic amblyopia are due largely to inaccuracies in field-taking. It is most difficult to map out a central scotoma, but with the modern refinements we will probably arrive at more uniform conclusions. The hand campimeter is an excellent instrument with which to pick out the scotomas, but he believes the Lloyd slate is probably a little more finished in working out the refinements. He believed that the differentiation which Dr. de Schweinitz made in reference to the peripapillary and ceco-central scotoma was very important. The ceco-central scotoma indicates involvement of the papillomacular bundle. Dr. Peter had never seen, in his studies, a single case of toxic amblyopia which was due solely to tobacco, although in England it is claimed that many cases of pure tobacco amblyopia without alcoholism are observed. In this country, however, he felt quite sure that few cases are seen in which there is not a history of alcoholism associated with the excessive use of tobacco, and now Dr. de Schweinitz has added his very interesting observation of the effects of diabetes when associated with the excessive use of tobacco in bringing about the same form of scotoma rather than a simple pericentral one.

Prolonged Miosis for the Control of Increased Intraocular Tension, with Remarks on the Preparation of Solutions of the Miotics

Dr. G. E. de Schweinitz and Dr. B. F. Baer, while expressing the conviction that an operation destined to produce a fistulous (filtering) area offered the best treatment of chronic glaucoma, and should, in suitable instances, be performed as early as possible nevertheless recognized that for various reasons operation could not invariably be performed, and that miotics must always maintain an important place in the therapeutics of glaucoma, both with and without the assistance of operative interference.

They quoted the case histories of patients with chronic glaucoma, whose eyes had maintained normal tension, vision and field of vision, solely under the influence of miotics, in one instance for over twenty years. Also, case histories indicating that one eye had remained in satisfactory condition, but the other eye had steadily lost ground, and other histories which showed that although miotics had availed for years, a period had arrived when they failed in this respect, and additional ones in which after a long period of satisfactory tension and vision conditions, sudden attacks of acute glaucoma had supervened, in one instance terminating in one eye in malignant glaucoma. All these facts showed plainly, they said, that it was not safe to trust to miotics alone, unless assured that the patients would remain under frequent expert observation—an assurance which only too often could not be secured. Hence their conviction that a suitable operation, in proper circumstances, was the best procedure.

One untoward action of the long-continued use of miotics consisted in the development of the well-known toxic conjunctivitis, although in some instances (cases here quoted) the drug had produced no deleterious conjunctival influence, even when used for years—in 3 cases for twelve, fourteen and sixteen years respectively.

They next described, by permission, the method of preparing miotic solutions (which would apply equally well to mydriatic solutions) devised by the well-known pharmaceutical chemist, Mr. J. W. England, who wrote as follows: "I have been making solutions of pilocarpine hydrochloride and physostigmine sulphate with a special rose-water, filtering through paper. The formula for the 'special rose-water' (No. 1) used is: Boric acid, 18 grains, and stronger rose-water, 4 fluidounces. Dissolve the boric acid

in the stronger rose-water and filter into a sterile bottle. If the rose-water is cloudy from supersaturation of volatile oil, mix with purified talc before filtration. To filter, use a glass funnel with a flattened pledget of absorbent cotton in the neck, and upon this rest the folded filter-paper. Return the first portions of the filtrate if not absolutely clear.

"There is something more in rose-water than the odor. It seems to be slightly anesthetic, however small the amount of oil in the rose-water may be. The chief constituents of oil of rose are the alcohols geraniol and citronellol, the two together being present to the extent of 70 to 75 per cent, while the citronellol constitutes about one-fourth of the liquid portion of the oil. Traces of esters of these alcohols are also present. (B. P. C. 1923, 765). I believe that this 'special rose-water' can be still further improved by adding 0.85 per cent of sodium chloride, making it a physiological salt solution in rose-water. The formula would be: Boric acid, 18 grains; sodium chloride, 16 grains; stronger rose-water, 4 fluid-ounces." Prepare as for "special rose-water No. 1."

This solution had been used by the author with perfect success, but only once, in a patient with chronic glaucoma, whose conjunctiva quickly became inflamed when the salts of pilocarpine or physostigmine was employed in water solution. They published, with his consent, Mr. England's formula in order that others might try it in suitable cases and thus sufficient information be gathered to establish, or not, its value as a substitute for the ordinary liquid solutions of miotics.

Some Interesting Changes in the Vessels of the Fundus

Dr. L. Waller Deichler and Dr. A. Barlow presented three cases of changes in the vessel of the fundus. The first: Mrs. M. W. B., aged forty-three years, was seen on December 12, 1917. The past history was negative. Ocular trouble began in August, 1911, when, within a period of a few hours, her vision was reduced to light perception. Slow improvement occurred, and in March, 1912, she had recovered two-thirds normal vision. In August, 1917, she was told that her vision was normal in that eye with correction. Shortly thereafter vision was again suddenly lost, accompanied by neuralgic pain. Injections of colloidal iodine were given over a period of two months. Vision, 15/200 in the right without glass;

15/100 with glass. In the left eye, 15/20 without, and 15/12 with glass. Examination of the right eyeground under mydriasis showed vitreous opacities, large chorioretinal disturbances from the disk over the entire macular region and well into the periphery, with atrophy and pigment-heaping. The choroidal circulation was plainly visible throughout this area. No hemorrhages were present. Large relative and absolute scotomas were present. The left eyeground showed retinal disturbances and two small, glistening spots in the choroid up and in from the disk. A relative scotoma was present. When seen in October, 1925, the condition was much the same, except for more pronounced perivasculitis of the choroidal vessels, which condition led to the present presentation and study.

The second case, seen and studied with Dr. Barlow: Mrs. E. S., aged fifty-four years, was first seen on November 13, 1925. She had lost the sight of the left eye four years previously through retinal hemorrhage, the result of high blood-pressure. Vision: Right, 20/30; left, fingers at 9 inches. Examination of right eye showed change of arteriosclerosis of the retinal vessels. Vitreous clear. A few small, linear hemorrhages are present on the temporal side of the disk. The left eye shows cloudiness of the retina and disk; hemorrhages and atrophic areas are present. The arteries show sclerosis and tortuosity, with perivasculitis and irregularity. The veins are unusually looped and tortuous. The ocular picture is the result of general arteriocapillary fibrosis with cardiac hypertrophy. Arterial tension varies from 218/120 to 246/136. Renal involvement is also manifest.

The third case, H. J., aged forty-six years, a woodworker, was first seen on January 20, 1926. Vision: O.D., 20/70 without correction; 20/40 with correction. O.S., 20/50 without correction; not improved with glass. The ophthalmoscope shows no lesion in the right eye. In the left is a fluid detachment of the retina above. Dense band of vitreous exudate extends transversely through the vitreous, upon the surface of which are several fine bloodvessels which, toward the extremity of the exudate, end in numerous fine and anastomosing loops, transforming the mass into a connective tissue structure. The disk edges are blurred and many small hemorrhages are scattered over the retina. When seen again on February 18, 1926, the condition of the left eye had decidedly changed. Many large and small floating vitreous opacities were present and the detachment had disappeared. A dense, pure-

white, glistening, irregular sphere occupied the center of the vitreous about one disk-diameter above the disk, from the margins of which an irregular fernlike growth with many fine anastomosing blood-vessels extended outward. On a second projection to the nasal side above and to the temporal side this same fernlike formation is shown, with a dense, white streak running well out to the periphery, with a distinct bloodvessel following the streak, and at its temporal end curving downward and backward to become continuous with the superior temporal artery.

An Intraocular Tumor of Unknown Pathology

Dr. Aaron Barlow's patient, Mrs. B. G., aged fifty years, came under observation at the Mt. Sinai Hospital on January 30, 1926, with a history of having fallen and struck the left eye three years previously. The consequent swelling disappeared, after the use of hot compresses, in about two weeks. Medical aid was not sought at that time. Later, however, she discovered that she could not see with this eye, and presented herself for examination on December 2, 1922, at which time vision in O.D. was 20/30; O.S., fingers at 6 inches. External examination was negative. Ophthalmoscopic examination of O.D. was negative; O.S. showed edema of the temporal portion of the retina above and extending downward to the macular region. Urinalysis was negative. She did not return for treatment but went elsewhere and had glasses prescribed.

Examination on January 30, 1926, showed vision O.D. 20/20; O.S., fingers at 3 feet eccentric. Inspection externally, negative. Ophthalmologically, O.D. negative. O.S. showed a linear opacity in the lens cortex at 7 o'clock. Through the upper part of the well-dilated pupil is seen an opaque, grayish body extending upward and outward and well forward into the vitreous. This is nearly spherical, sharply circumscribed, about the size of a large myopic disk. Its margins are everywhere free, so that the whole mass appears to be suspended in the center of the vitreous midway between the lens and the disk, without any visible attachments. Above and to the temporal side about one-third of the disk is obscured by the mass. The remainder of the disk is plainly visible, with fairly well-defined margins and normal bloodvessels. The anterior surface of the mass presents an abundant supply of fine

bloodvessels which are lost at the margins. The fundus, excepting the part hidden from view, presents no abnormalities.

There are numerous fine, floating, vitreous opacities, and a few filaments. To the inner side of the mass there is a small cone-shaped, fixed vitreous opacity with a fine, short, undulating filament attached to its base. Transillumination reveals a translucent mass with thickened capsule. Slitlamp study shows the capsule with brilliant white criss-cross network of fibrous tissue. The Wassermann is negative.

Discussion. Dr. H. O. Sloane stated that, through the courtesy of Dr. Barlow, he had the opportunity of studying this case at the Mt. Sinai Hospital. This patient had a body somewhat larger than the disk, whitish in color, with its surface covered with fine vessels (apparently arteries) which seemed to lie in front of the retina and apparently quite free in the vitreous. Its margins were completely rounded and in getting the patient to move her eye in all directions all the edges could be distinctly seen. Dr. Barlow stated that there was an attachment to the retina below and to the nasal side, but at the time of Dr. Sloane's examination he did not see it. It was his opinion that the new-growth was probably cystic in nature. The external appearance of the eye was normal and the tension taken by the finger was normal.

MARCH 18

Case of Drusen of the Fundus

Dr. Hunter W. Scarlett showed a boy, aged nine years, who presented, in the right eye, a translucent, glistening, cone-shaped mass down and out a short distance from the nerve head, extending into the vitreous about three diopters.

The usual location of these lesions, according to Fuchs, is in the periphery, but they may be found in the papillomacular region.

These excrescences are supposed to arise from the lamina vitrea and are seen mostly in the old, although they may occur in the adult and even in the young.

Case of Xeroderma Pigmentosa

Dr. Burton Chance exhibited a case of xeroderma pigmentosa in a young man of seventeen. The disease, which appeared at two years of age, has spread over the upper half of the body, the

hands and forearms, into the scalp and has seriously involved the eyelids and globes. The lids showed cicatricial changes and ectropion, that of the left eye being quite marked; the conjunctivas were atrophic and so shrunken as to contract the fissures. The scleras were freckled, quite early, and the corneas became ulcerated, the right being hazy, and leukomata mark the left. The eyes are sensitive to light and so tender to the touch that it is with difficulty that the globes can be inspected. The ectropion of the left has been greatly reduced by recent electrocautery puncturing of the conjunctival surface. This is the second case so afflicted in the family; the elder having died from the extension of the disease. The parents are Jewish, full cousins; but in no member of the family on either side has there been any history of the disease.

A Memoir of Dr. S. Lewis Ziegler

BY DR. PAUL J. SARTAIN

Dr. S. Lewis Ziegler, a Fellow of this Section since 1899, and chairman of the Section in 1915 and 1916, succumbed to an attack of pneumonia on January 4, 1926.

Dr. Ziegler was born in Lewisburg, Pa., February 5, 1861; received his preliminary schooling there and then entered Bucknell University, from which he was graduated with the degree of A.B. in 1880.

He came to Philadelphia in 1882, and matriculated in the medical school of the University of Pennsylvania, and was graduated in 1885, then serving as intern at the Germantown and Episcopal hospitals, and finally at the Wills Eye Hospital, in which he later became attending surgeon and subsequently executive medical officer. In 1899, he organized the eye clinic at St. Joseph's Hospital, and remained a member of the staff up to the time of his death. In recognition of his work, he subsequently received his M.A. from Bucknell, and had the honorary degrees of Sc.D. and LL.D. conferred on him by Bucknell and Lafayette respectively.

A close observer, of keen and analytical mind, he studied his cases with an untiring exactitude which generally brought him to conclusions logically correct and sometimes at variance with the usually accepted theories of etiology and pathology. His treatment, always rational and often original for difficult and obscure cases, has in many instances become accepted as most efficacious. His ingenuity in devising and supervising the making of new

instruments in ophthalmic practice was of absorbing interest and genuine pleasure to him, and some of his special operations have received well-merited international recognition and adoption. As an operator, skilful, decisive, quick, meeting the emergency as it arose, often anticipating a complication, his results averaged high, and in many cases referred to him after unsuccessful operations his V-shaped iridotomy, which he so dextrously performed, gave brilliant results.

Dr. Ziegler took the keenest interest in the new developments in the general field of medicine, and his activities outside his own specialty were evidenced by his valuable work in the local chapter of the American Red Cross, and his successful administration as Director of the Bureau of Health and Charities of the City of Philadelphia.

For some years Dr. Ziegler had been writing and collecting material for a monumental work upon the surgery of the eye. He spent much time in the great libraries of Europe consulting original authorities and having photographic copies made of the portraits of the most noted ophthalmic surgeons from the earliest times. These portraits, together with reproductions of an admirable set of drawings of operations made under his direction, would have been the illustrations for the text, still unfinished, which it was the ambition of his life to complete.

His appreciation of art and love of the beautiful led him into the field of collecting during his visits to Europe and made a pleasant diversion from his work; relaxation he had never learned.

The interests of the College he had always at heart and was ever ready to serve it to the best of his ability. His genial personality, sterling honesty in his work and unswerving loyalty to his friends remain with us as a cherished memory.

Angioid Streaks of the Retina

Dr. J. Milton Griscom exhibited a case of angioid streaks of the retina corresponding in most details with the cases already reported. Discussion of the condition and an exact description of the fundus picture was postponed until a later date.

Discussion. Dr. Zentmayer showed the plate of a case exhibited before the Section several years previously. He said he had expressed the opinion that the lines were pigmented congenital vessels. The theory which seems

best to explain this characteristic type of pigment streaks is the one advanced by Collins, that they are the posteiliary vessels rendered visible by the entrance into their perivascular lymph spaces of hemosiderin. There are other types of pigment lines in the retina, and in one of these both Alt and Fuchs have found them to be due to folds in the pigment epithelium.

Dr. G. E. de Schweinitz exhibited some water-colors illustrating angioid streaks in the retina, the case histories having been reported a good many years ago. The lesions exactly corresponded with those observed in all other cases of this affection, and were especially interesting in that they occurred in two members of one family –brothers –indicating in this instance a familial tendency. Dr. de Schweinitz had originally adopted the explanation advanced by Lister that they, the streaks, represented newly formed vessels which had penetrated inflamed tissues, along which pigment deposits are arranged. But since Coates's suggestion that the streaks arise along vessels which belong to the choroid rather than the retina, and Treacher Collins's observations that they are due to "hemotogenous pigment from subchoroidal hemorrhages deposited in the perivascular spaces around the short ciliary arteries, especially in the neighborhood of the disk and circle of Zinn," he believed that a more satisfactory explanation of the production of this condition had been advanced.

Dr. Burton Chance asked whether the patient was a negro. He could not recall having seen a case showing such streaks in a colored person, but had found them in those of fair complexion. Might not this case be one of excessive pigmentation without inflammatory or degenerative changes in the retina?

Dr. Edward A. Shumway said that, so far as he knew, angioid streaks in the retina had not been reported in colored patients, and a new factor of pigmentation might be introduced in the present instance which might represent a deposit of pigment along a deeper set of vessels without a previous hemorrhage into the retina.

Case of Disciform Keratitis

Dr. William Zentmayer exhibited a classical case of disciform keratitis in a young man, a student. The lesion was slightly eccentric and had a diameter of 8 mm. There was a characteristic, dense margin. The central area was less dense and showed dot-and-line opacities. The ciliary congestion was moderate.

The condition had existed for seven weeks, and began with the sensation of a foreign body. There was no history of injury. The tonsils are infected.

Discussion. Dr. G. E. de Schweinitz expressed his satisfaction that Dr. Zentmayer had brought for observation his patient, who typically exhibited a real keratitis disciformis, that is, a ringlike or disclike keratitis, which corresponded in all respects with Fuchs's original description.

He deprecated the casual use of the term "disciform keratitis," as it so frequently occurs in literature, in many instances the lesion being simply dislike in appearance, but in no way conforming to the type of keratitis as recorded by Fuchs. He was aware that certain authors – for example, Verhoeff – were unwilling, from the etiologic standpoint, to admit that this corneal affection should be regarded as a clinical entity. But nevertheless, he believed that "disciform keratitis" should be used to express a definite conception, that is, to describe an individual type of keratitis.

Exhibition of Two Cases of Nodular Degeneration of the Cornea

Dr. William Zentmayer showed 2 cases, sisters, who were referred to the Wills Hospital by Dr. D. Fordyce in 1922. E. R., aged nineteen years. Poor vision in the left eye since eight years of age. Vision: R.E., 6/30; L.E., 1/60. In both corneæ there were subepithelial, grayish, tortuous, thick lines. The overlying epithelium was shagreened. The corneæ were somewhat conical. No detailed view of the fundus was obtainable. March 16, 1926, vision was: R.E., 2/60; L.E., 1/60.

C. R., aged twenty-eight years. History of mild inflammation in both eyes when five years of age. R.E., grayish, bread-crumb opacities, very dense over the central three-fourths of the cornea; periphery nebulous. L.E., typical nodular opacities in Bowman's membrane. Surface of the cornea shagreened.

March 16, 1926. The opacities in R.E. have coalesced and the central three-fourths of the cornea has a dense, almost uniform, gray appearance though there are still some subepithelial nodes lifting up the epithelium. The perforating vessels are greatly enlarged and tortuous. Vision in each eye reduced to hand movements.

In 1922, this case was treated by application of the thermophore at a temperature of 140°. This caused some exfoliation of the epithelium, with a slight improvement in the vision. This was but temporary. Both cases received thyroid extract. Physical examination was refused by the patients.

Mention was made of a family in which nodular degeneration of the cornea was present in nine members of the family, thirty-six having been examined. Two other members, not examined, were said to have been similarly affected.

No distinctive changes were noted in the corneal nerves with the slitlamp.

Discussion. Dr. de Schweinitz, referring to the treatment of such cases as Dr. Zentmayer has reported, described his efforts to relieve a typical case of this character. Remembering Wehrli's contention that the disease represented a form of chronic tuberculous disease of the anterior layers of the cornea, he had tried prolonged tuberculin treatment, associated with persistent massage of the cornea, with the aid of dionin and yellow oxide salve, but without the slightest benefit. Thus far, it would seem, no therapeutic measure had been devised which yielded any satisfactory results.

Dr. Burton Chance said these eyes recalled to his recollection those of a man and his son whose history he had reported several years ago at a meeting of the Ophthalmological Society. His experiences had been quite like those of Dr. de Schweinitz. All sorts of examinations were made, and every kind of remedy proposed was employed, yet inquiries into the causation of the affection and attempts at the lessening of it were without avail.

A Case of Microphthalmus with Orbital Cyst

Dr. E. A. Shumway and Dr. A. R. Renninger reported a case. P. A., aged fifteen months, female, who was brought to the Children's Hospital Dispensary on March 27, 1925, and was seen by Dr. Renninger. The child was born December 5, 1924, and weighed 6 pounds 3 ounces at birth. It was a well-developed infant and has been in good health since then except for a recent attack of bronchopneumonia, for which it was treated at St. Agnes Hospital. Here it came under the observation of Dr. George F. Kelly, who has been good enough to send us some details of the family history. The condition of the left eye was present at birth and the mother thinks the cyst has increased in size. The father of the child is living, but has some heart affection and a chronic cough. The mother had chorea at the age of twelve years, but is now comparatively well. There were five children; the oldest is eight years of age, is deaf and is in a home for the deaf; the second, six years old, is very nervous; the third was stillborn at about six and a half months; the fourth, now four years old, has a bad heart, and the present child is the fifth.

She has been examined also at the Wills Hospital, Methodist Hospital, and by a number of other physicians. She was brought to the Children's Hospital from March to September, 1925, and during this time Dr. Renninger could detect very little difference in the size of the cyst.

Present Condition. The anterior part of the orbit is occupied by a large, thin-walled cyst, which bulges forward between the eyelids and seems to be divided by septa in its interior. It transmits

light and is freely movable. On its lower surface, well back in the orbit and so hidden by the cyst that it at first escapes notice, is a rudimentary or microphthalmic eye which is connected with the cyst and moves with it. The cornea is turned forward, is about 4 mm. in diameter, and the iris shows a coloboma below, apparently at the opposite side from the connection with the cyst. No other congenital abnormality is present. The fellow-eye is normal and shows no evidence of coloboma. *X*-ray examination of the cyst and orbit at St. Agnes Hospital was negative.

Cyst of the orbit in association with microphthalmus is a comparatively rare condition. According to Parsons, it was first accurately described by Arlt in 1858, as the results of two anatomical examinations by Wallman. According to von Hippel, Jr., in Jena, who wrote the section on congenital anomalies in the second edition of Graefe-Saemisch, and in Schwalbe's *System on Morphology and Malformations of Man and Animal,* 82 cases had been reported up to 1908, 26 with anatomical examination of the eyeball, in connection with the cyst. These examinations have proved positively that the cysts stand in direct relation to the development of the eyeball, with which they are in direct connection, and whose constituent parts are found also in the cyst. Every gradation may be met with, from the ectatic coloboma of the choroid to the fully developed cyst.

There are two chief theories as to the origin of these cysts, associated respectively with the names of Arlt and Kundrat: Arlt's theory is that the cyst is due to extreme development of ectasia in the neighborhood of a defectively closed cleft, in the *secondary* optic vesicle. Kundrat's theory is that the cyst is formed from the noninvaginated *primary* optic vesicle; he assumes that the secondary optic vesicle does not develop, but that the distal part of the primary optic vesicle grows into the surrounding mesoderm and develops into a cyst. This theory may account for the cases in which complete anophthalmus is found, but where there is microphthalmus, which is the usual condition, Arlt's theory is the generally accepted one.

The ocular cleft, through which the bloodvessels pass into the interior of the secondary optic vesicle, closes early in fetal life, cutting off the mesoblastic structures external to the secondary optic vesicle from those internal to it. Occasionally a union persists between these mesoblastic tissues inside and outside the retina, or there may be a delay in the separation so that there is

a defective closure of the gap in the wall of the vesicle, and a coloboma results. Eyes with this form of coloboma usually do not expand to the full extent and are microphthalmic. Moreover, the region in which a coloboma occurs is frequently a weak spot in the walls of the globe and is very liable to become ectatic, sometimes to such an extent as to produce these large cystic protrusions.

Microscopically, the cysts have a fibrous tissue wall, derived from the sclerotic; but this, as in our case, may be absent. The choroid is absent over the cyst wall or extends only a short distance into it. The cysts communicate with the interior of the globe by an opening, which is usually small, but varies in size. They are lined by representatives of one or both layers of the secondary optic vesicle, the outer one of which develops normally into the pigment epithelium of the retina, while the inner represents the retina proper. In the cyst the pigment epithelium is much altered, forming a somewhat laminated layer of cellular tissue. The retina proper is folded near the orifice, but also lines the cyst, and it may be well developed, so that the layers are distinguishable. In some cases it lies as in the eye, while in others it has a so-called inverted position, *i. e.*, the rods and cones are turned toward the lumen of the cyst. As a rule, these cysts are connected with the under surface of the eyeball, but occasionally they are found above and appear beneath the upper lid. For these cases, Ginsberg has advanced the hypothesis that the cysts are due to nipping off a knuckle of the secondary optic vesicle by mesoblast, so that it becomes incarcerated, though still in connection with the eye, and under the intraocular pressure it expands. This theory would explain cysts in any position.

Motais Operation for Ptosis

Dr. Charles R. Heed exhibited a young woman, aged eighteen years, who had congenital ptosis of the left eye and upon whom he had recently performed a Motais operation with the modification as suggested by Dr. William T. Shoemaker.

In the latter part of the operation, as described by Motais, the tongue of the superior rectus tendon is drawn downward into a pocket between the anterior surface of the tarsus and the skin by passing the two needles into this blind opening and bringing them out through the skin, 2 mm. from the lid margin, about 4 mm. apart, and tying over a roll of gauze. There is some uncertainty as to the position of the tongue, and not infrequently the suture cuts through

the tendon before adhesions have taken place. Dr. Heed recorded three failures due to slipping of the tongue from imperfect attachment.

In Dr. Shoemaker's method, a small incision is made through the skin of the lid midway between the superior tarsal border and the lid margin. The tarsus is exposed to view and a small incision passes through the conjunctiva just above the superior margin. The two needles, with the suture and tendon, are brought through this opening. A firm attachment is then made by passing the needles into the tarsus near its superior margin. The needles are then brought out through the skin near the lid margin and tied over a roll of gauze. Two sutures close the skin incision. The modification enables one to view clearly the position of the tongue, as he firmly attaches it to the tarsus, and does not complicate the operation.

Dr. J. H. Dewey exhibited a case of Motais operation for ptosis which showed some overcorrection of the ptosis, which is rather in contradiction to the generally accepted opinion that there is no danger in obtaining such a result. The overcorrection is no doubt due to bringing the suture too near to the ciliary margin of the lid. He uses the open operation, and after tying the suture through the skin below the line of incision, with the tongue of the muscle fully exposed to view, he attaches the muscle with a second suture through the skin above the line of incision. He believes that this greatly reduces the change of failure.

Discussion. Dr. Zentmayer said he regretted not having brought a recent Motais operation to show, as there would have then been an ideal result, an overcorrection and a failure. He had done the open operation and had followed O'Connor's suggestion of shaving off a layer of the sclerotic with the tendon tongue. Despite this, the suture tore out, due to the operator making entirely too much traction on the sutures after bringing them out at the border of the lid. Forty-eight hours later, under ether, the tongue was found and stitched firmly to the surface of the tarsus, and the result now bids fair to be a success.

Dr. William T. Shoemaker called attention to two modifications of the original operation which he published in 1907 (*Annals of Ophthalmology*, October, 1907). The first was the open method which Dr. Heed practised in his case, and which he found easier of execution, more accurate and more satisfactory. The second modification which was suggested, but which, so far as Dr. Shoemaker knows, has not been tried, is based on his conviction that in the final analysis any Motais operation can do nothing more than physically attach the base of the eyelid to the eyeball at or near the insertion of the superior rectus muscle. If this is true, it is quite unnecessary

to laboriously prepare a tongue of rectus muscle to carry into the lid tissues. The same thing can be accomplished by exposing the rectus muscle and the upper margin of the tarsus and, without cutting or interfering with the tendon, pass a double-armed suture beneath the middle half, third or any other portion of it, and then through the tarsal margin and on to the skin surface of the lid and tie. This would bring the base of the lid in firm contact with the eyeball at the desired point and the two wounds would unite. This is the very valuable Motais principle, and it is difficult to see how the original technic can do more, with the tissues in question. The piece of prepared rectus tendon is inextensible and cannot be carried independently into the lid, but the lid must be carried to it, and there unite at the original site of the tendon.

APRIL 15

Osteoma of the Orbit

Dr. E. B. Miller exhibited a case of osteoma of the orbit, with the following history: C. F. J., aged forty-six years, electrotype worker by occupation, had always been well until July 7, 1925, when he became nervous, had difficulty with speech and loss of memory. He was taken to the Frankford Hospital, where he remained for two weeks; he gradually became better, recovered his memory and was taken to Atlantic City for five weeks. No other treatment was given. He was taken to the University of Pennsylvania, and seen by Dr. Spiller, but not examined. He was later given a note to the Philadelphia Hospital, but when he saw the building he said it looked like a prison and refused to enter. Following this he went to a physician for some trouble with his nose and was referred to the Polyclinic Hospital for *x*-ray study.

At the time that Dr. Miller saw him, he had not been working for some time because of a supposed nervous breakdown. While cutting-up and laughing, he became suddenly blind in his left eye. For some time before this he had noted that his sense of smell and taste was considerably disturbed. He had been wearing glasses for work; O.D., +0.37 sph.; O.S., +0.25 sph. +0.25 cyl. ax. 90 degrees, with +1 sph. added for near. Vision without glasses was 20/20 in each eye. Fields taken by the hand method disclosed left homonymous hemianopsia. This was confirmed by a careful study on the campimeter. The right blind spot, studied on the Lloyd slate, was enlarged on the temporal side. External examination

was entirely negative. Fundus examination showed oval, slightly filled-in disks, with narrow, slightly tortuous vessels.

On the right side of his head, in the temporal region, was a bony tumor. The *x*-ray disclosed the continuation of this bony mass into the skull cavity. The mass itself was hollow, 8 cm. in diameter, almost filling the right temporal fossa. There was also a bony growth in the region of the sella.

Neurological examination and all laboratory tests were negative. He is left-handed and his speech center is probably on the right side, which account for the speech disturbance.

Discussion. Dr. Emory Hill, of Richmond, Va., considered this case especially interesting because of the significance of such bony tumors of the skull as recognized nowadays by the neurologic surgeons. He stated that several of them had been seen in Richmond, and it had been confidently expected to find an endothelioma beneath the bony mass. He said that this man, unless he proves an exception to the rule, will show a large but benign and encapsulated tumor, which can be removed with recovery from his disabilities. The ordinary excision of the bony tumor would be a very bloody procedure and useless, because it would not reach the essential lesion; on the contrary, complete enucleation of the intracranial tumor, with adequate hemostasis, as practised by the neurosurgeon, will almost certainly effect a cure.

Case of Obstruction of a Branch of a Retinal Artery

Dr. Warren S. Reese presented a case of this sort in a woman, aged twenty-six years, who, two years ago, had an attack of dizziness accompanied by marked reduction of vision in the right eye. This cleared up in a few seconds, but a permanent defect in the right upper field remained. There was a second similar attack a year later, following which the eye returned to its former condition.

There is a loss of the entire right upper field, extending almost down to fixation. The ophthalmoscope shows a marked reduction in caliber of the inferior temporal and nasal arteries in the right eye, and a perivasculitis along each of these vessels. Both these vessels come off the disk separately, although they diverge as though coming from a common trunk.

The only significant thing in the general examination is that each of the abovementioned attacks was preceded by an infection. About a month previous to the first attack the patient had a mouth infection and was told by her dentist that she had abscesses. Some time previous to the second attack the patient had quinsy.

Complicated Cataract following Injury

Dr. Warren S. Reese presented the case of a male, aged thirty years, who was struck in the right eye five weeks previously with a stone. The eye had been perfectly well previous to the accident.

The right eye shows a linear scar of the cornea up and out. Behind this is a rupture in the pupillary edge of the iris and a posterior synechia. Back of this are some streaky opacities such as one sees in a localized lens injury. In the cortex of the center of the lens is a star-shaped opacity described by Fuchs as a complicated cataract.

The case is of interest from the standpoint of whether the cataract is really traumatic or (and this seems more likely) whether it is due to uveal disease induced or lighted up by the injury. The *x*-ray is negative.

The Todd Muscle-tuck, with a Modification

Dr. Frank C. Parker, in presenting a paper upon this subject, said that any resection of an ocular muscle is associated with a certain element of uncertainty unless the holding stitch is inserted through the scleral tissue, the latter being split by the needle. This closely approaches the danger zone. Aside from this, should anything unforseen occur and the muscle slips its stitch, conditions are necessarily complicated.

No stitch inserted into the conjunctiva or episcleral tissues alone can be depended upon to hold a shortened muscle *in situ*. They invariably pull out in a few days and the effect of the operation is lost. Any further straightening of a squinting eye dependant upon the drawing together of the closing conjunctival sutures may be disregarded in the final result.

Owing to the dangers associated with the scleral stitching, together with the necessity of removing a section of the muscle or tendon, the Todd tuck, with a modification, was selected. This requires no scleral stitching. No muscle or tendon is removed. Should anything unforseen occur, the preoperative status is not impaired.

The modification consists of inserting the muscle-tying stitch beneath the tucker arm instead of over the arm, and in a thorough removal of all fascia surrounding muscle and tendon for a distance of about 16 mm. by means of a cup-shaped, saw-toothed curette. Also, the tucker arms have been changed to an angle of 45 degrees

and the closing conjunctival stitch has been altered, this latter, however, being considered unimportant and acting simply as a means of closing the wound and allowing the flat approximation of the tuck to the sclera.

A careful tenotomy of the opposing muscle is done before the tuck is made. This tenotomy should not be of the free, slashing order, but confined strictly to the tendinous insertion and not beyond its limits. Small snips of the scissors opened for only a few millimeters are employed.

In doing this modified tuck, the only cut made is the original opening through the conjunctiva and capsule. No further cutting is done to expose the muscle, this latter being accomplished by means of the closed blades of the scissors, which are used to push back all tissue, giving a clean exposed muscle.

The writer, in ten years' use of this method, has never experienced an untoward result as to sloughing or other pathological change. The operation seems to be perfectly safe.

In tying off the tuck, No. 1 plain gut is employed. Chromacized gut is inadvisable.

From photographs shown, the operation is capable of correcting the highest degrees of squint.

This modified tucking operation must be done with care and proper regard for the described technic in order to secure the desired result.

The Action of Atropine in Acute Inflammations of the Eye

Dr. Francis Heed Adler pointed out that the explanation of the action of atropine in acute inflammations of the eye, as given in text-books, is that it paralyzes the endings of the third nerve and thereby puts the ciliary and sphincter muscles at rest—paralyzing accommodation and dilating the pupil.

The dilatation of the pupil is undoubtedly an important factor in the prevention of the formation of posterior synechia, but cases are often seen in which the formation of synechia is a rare occurrence and yet atropine is of decided benefit. This raises the question whether there is another action of atropine besides those enumerated. In some experiments previously published (*Archives of Ophthalmology*, Vol. 14, 1925, page 265) evidence was obtained which indicated that atropine had a direct effect on the ocular blood-

vessels, decreasing their permeability to protein molecules. If atropine has such an action, it would explain one of the effects of this drug in ocular inflammations, for one of the cardinal signs of any inflammatory process is a leakage of protein from bloodvessels. Any drug which tends to prevent such a leakage will allay inflammation.

A series of experiments were devised to test this hypothesis. Dionin, subconjunctival injections of hypertonic salt solution, and oil of mustard were found to increase the protein content in cats' eyes. Quantitative determinations of the protein content were made in a series of eyes in which these drugs had been instilled, both with and without a previous atropinization of the eye. It was found that atropine definitely checked the increase in protein caused by these drugs, and the experiments were so controlled that the conclusion could be drawn that atropine did have a direct action on bloodvessels, decreasing their permeability to protein. For methods and experimental data the original article should be consulted.

Discussion. Dr. H. Maxwell Langdon said that, according to Dr. Adler's findings, it would seem that in using both atropine and dionin in inflammatory diseases of the eye we are doing a contradictory thing, and yet I think it is common usage to do so, the atropine tending to lessen the formation of inflammatory products, whereas the dionin would seem most surely to increase it. Would it not be better then, except in cases where there was severe pain, to withhold the dionin until later stages of such conditions?

Dr. Edward A. Shumway said that Dr. Adler's results, showing the effects of atropine on the bloodvessels and the reduction of the protein output, and the opposite effect of eserine, have a very practical value in the treatment of ulcers of the cornea and other inflammatory conditions. Many of the members have probably seen hemorrhages from the iris into the anterior chamber, in secondary glaucoma, in old cases of iridocyclitis, following the use of eserine, which aggravated the condition. In some text-books eserine was advised in ulcer of the cornea, when there was danger of perforation in the periphery, but this treatment had always seemed to him unwise because of the increase in the hemorrhagic tendency, and atropine should be the better drug in any ulcer, whatever its position.

Sarcoma of the Choroid

Dr. Burton Chance reported 2 cases of small, round-cell sarcoma of the choroid which had come under his observation, the only ones he had received in the past ten years. The first was in a physician

who, when seen, was in violent, fulminating glaucoma, and evidently profoundly affected systemically. The patient died after three years from widespread involvement. The other, under observation at the same time, is alive and apparently in perfect health. In the first case there was orbital involvement, the orbit, however, could not be treated by radium until several weeks after enucleation. In the course of time the orbit disease was presumably entirely arrested. The other being a hospital patient, escaped all attention immediately after enucleation. He has recently returned after three years, but as his general health has been robust no radiations have been applied at all.

Many questions are still unsolved concerning the malignancy of ocular tumors. It is seldom that cases can be followed in the subsequent years satisfactorily by the ophthalmic surgeon. Chance wonders whether the tumors with much pigment, irrespective of the character of the cells, are less metastatic than those with but little pigment. In the sections of the fatal case the pigmentaton. seemed to be so arranged as though it were flowing out of the tumor, whereas that of the surviving one was so dense as to obscure the cells in many sections. Two thoughts he holds: First, that when glaucoma sets in, enucleation must be performed without the slightest delay; the other is to apply radium as speedily as possible, and for as long as can be and might be, to the orbit.

Discussion. Dr. Edward A. Shumway said that he thought the type of cell of the tumor was of more importance than the presence of pigment in the production of metastasis. Round-celled growths were more likely to spread rapidly than spindle-cell types or the endotheliomas.

Orbital Swelling Associated with Chronic Frontal Sinusitis (Previously Diagnosed as Osteoma), with Sudden Extensive Edema of Upper Face and all Four Eyelids, and with Suggestive Ocular Fundus Changes

Dr. G. Oram Ring reported a case of frontal sinusitis with suggestive ocular fundus changes.

Mrs. T., aged fifty-eight years, was referred to him for operation March 21, 1922. Previous diagnosis, osteoma of the upper, inner angle of right eye. This case presented a contrasting picture to one presented by Dr. William C. Posey, a pioneer in the work in the present century.

The patient had a swelling in the inner-upper angle of the right orbital region quiescent for nine months. This all changed in forty-eight hours to a fulminating edema of the upper face and all four eyelids. High temperature, 104°. Initial examination showed a circumscribed swelling of the upper-inner orbital region, the size of a hickory nut, extending to the inner third of the right upper orbital rim, and downward to the root of the nose. The fissure was about one-third normal. The eyeball was deflected down and out, and any effort at upward movement accentuated the divergence. Diplopia was present. Vision, 20/40. The nasal half of the nerve was blurred. The field was restricted on the temporal side. Moderate Van de Hoeve scotoma was present. *X*-ray report and the interpretation given as orbital lipoma with negative frontal sinus findings. Intranasal study showed a deflected septum, obscuring the middle turbinal and necessarily impairing frontal drainage. Sinus illumination negative. These examinations were made March 21 and 22, 1922. On March 23, forty-eight hours after the initial examination, the patient noticed increase of edema of the lids, associated with pain in the eyeball, with flushing. Symptoms increased in twenty-four hours, demanding immediate operation. The fulminating edema involving the entire upper face completely closed all four eyelids, and the swelling extended up on the forehead across the nose and down each cheek. Temperature, 104°.

Dr. B. A. Randall, in consultation, advised immediate operation and believed there was involvement of the frontal sinus.

Following incision along the upper two-thirds of the orbital rim, a large amount of pus was found. Further examination revealed an opening into the frontal sinus filled with granulation tissue. This was curetted and an extensive gauze drain was inserted. The wound healed in two months.

X-ray examination by Dr. W. F. Manges, May 27, 1922, revealed no free pus in any accessory sinus.

The association of the writer with the case history narrated is the entirely simple one of merely opening, curetting and draining a diseased cavity, but it has rather a wider interest in that in its elaboration it overlaps the ocular, nasal, *x*-ray and the anatomical group study.

Four years have elapsed and the patient has suffered no further trouble. Vision, 6/7.5 full. Eye in a normal position.

Case History Citation. 1. Rapidity of orbital swelling, quiescent for a long period and then assuming a dangerous aspect.

2. Emphasizing clinical, over *x*-ray, study.

3. Van de Hoeve scotoma, without any demonstrable evidence of disease of the posterior paranasal sinuses.

4. Preference of a simple surgical practice in a more elaborate nasal drainage route.

5. Suggesting early rhinological examination, giving patients a chance for early submucous resection and draining frontal sinus by intranasal route.

Discussion. Dr. B. Alexander Randall said: "The admirable presentation of his case by Dr. Ring leaves little to be said with regard to it; so I will only endeavor to cast a sidelight upon the matter by citing a case of sinusitis seen nearly forty years ago that has been, in my experience, unique. The clinical picture of one stage of his condition is here shown; while photographs of horizontal sections of the frozen head, which I made some years ago at the Wister Institute, may aid in presenting the anatomical relations of these paranasal sinuses to the eye, eyelids and orbit.

Richard C., aged thirty-one years, white, employed at League Island, came to me at the Episcopal Hospital in 1889 suffering with severe headaches of some four months' standing, which had begun with a stuffy nose. He had been treated by advertising "catarrh specialists," but with increasing disability and suffering; when his eyes became involved, he had been told to consult an oculist. Ptosis almost closed his left eye, which was decidedly protruded, but fairly covered by the brawny eyelids; there was little conjunctivitis; vision, 6/12(?) in each, with P.P. 6 inches; there was some chemosis and upward deviation of the left eye and diplopia when the slightly dilated pupil was uncovered by lifting the rather rigid eyelid. Other ocular movements were poor, but without notable change as to the vertical diplopia present all the time when the eyes were open. The eye-grounds were nearly normal, although venous congestion was rather greater on the left. All specific history was denied but iodide and bichloride were given, with decided increase of his pain. Little seemed amiss in his nares, and consultation with Harrison Allen brought little light as to the exact seat of his trouble, although high scarification gave some relief. The diagnosis of sphenoid empyema seemed verified by a spontaneous gush of reddish mucopus a little later, with marked and progressive relief.

The unique feature of the case was the successive paresis of each external muscle of each eye, beginning with the inferior rectus of the left, then the right, and involving in clockwise sequence each other rectus. The ptosis seemed largely ascribable to the massive induration of the lids. The practically specific action of atropia on the accessory nasal sinuses had not then been grasped, nor the probing of the spenoid openings or infundibulum; so the case was not treated, as I would now hope successfully to do, with little delay. Neither grippe nor lues seemed to be assignable as the cause,

and the therapeutic test of the iodide making the condition decidedly worse was much more convincing of the absence of syphilis than the emphatic denial of the patient as to any known exposure. The problem of the exact anatomical localization of the sinus involvement can be only hypothetically and variously explained. To Harrison Allen, as to me, it was an enigma as yet unanswered.

Lesson to be Learned from Dr. Mitchell's Paper on "Eyestrain as a Cause of Headache" (Published in April, 1876)

Dr. H. Maxwell Langdon read a paper upon this subject in which he said that if a study of the medical literature is made, occasional references will be found to the fact that, in certain cases, ocular trouble had been found to be the cause of headache. This was not stated in any definite, conclusive way, however, until, in the August issue of the *Medical and Surgical Reporter*, 1874, S. Weir Mitchell, in a paper on the various causes of headache, included ocular strain. Apparently this attracted very little attention, and it was not until nearly two years later, in April, 1876, to be exact, that another article of his, published in the *American Journal of the American Sciences*, was convincing enough to arouse general medical interest, and therefore this month of April, 1926, may be considered as the semicentennial of the publication of this important fact.

In the *American Journal*, Dr. Mitchell made the following definite statements: (1) That there are many headaches which are due to disorders of the refraction or accommodation apparatus of the eye. (2) That in these instances the brain symptom is often the most prominent and sometimes the sole prominent symptom of the eye troubles, and that while there may be no pain or sense of fatigue in the eye, the strain with which it is used may be interpreted solely by occipital or frontal headache. (3) That the long-continuance of eye troubles may be the unsuspected source of insomnia, vertigo, nausea and general failing of health. (4) That in many cases the eye trouble becomes suddenly mischievous, owing to the failure of the general health or to increased sensitiveness of the brain from moral and mental causes.

Anyone who has had much to do with ophthalmology or neurology, I think, will agree that these brief paragraphs contain the gist of our knowledge of eyestrain, and that the best any later writer could attempt to do would be to expand and illustrate them, but that little essential material was left to be added to them.

Since this brief but complete paper, whole books have been written on the subject—much that was sound and sensible, and as much if not more that was silly. Writers familiar to all who read this made claims as to the terrible and wholesale troubles caused by eyestrain that were so extravagant that the whole subject became almost ridiculous, and, indeed, was ridiculed by physicians not conversant with the facts. Some of these writers claimed so much that the whole subject was looked upon askance, and accurate facts and statements disbelieved and disregarded.

In considering the subject, it must be kept in mind that we are working in that very flexible, fluid material of functional conditions, where we have great variation not only in the powers of different individuals, but also great variance at different times and under different conditions in the powers of the same individual, and that therefore it is not like weighing or measuring some tangible thing. An error of refraction which will cause much discomfort of various sorts in one person will, in a subject with a more stable make-up, produce no symptoms worthy of notice. This, to the uninitiated, is bound to be confusing and to throw a shadow of doubt over the whole matter.

Another reason for the skeptical attitude of some medical men is the inaccuracy of some refractions. One of the things most positively stated by Dr. Mitchell, and by Norris, Thomson and Dyer, the ophthalmologists on whose work his observations were based, was the need of careful and accurate correction of the defects *after the use of a cycloplegic;* many ophthalmologists are today throwing away the results of this work in their anxiety to gratify the haste of patients who naturally wish to avoid the annoyance of a cycloplegic; and some are pandering to this mistaken idea of the public in the fancied necessity, as one physician expressed it last summer, "to compete with optometrists." He was at once shown the error of this idea by a colleague, who said he "did not compete with optometrists but tried to do much better work." When a man known as a leading ophthalmologist will make such a statement as that on the floor of a medical gathering, it is not to be wondered at that lay people go astray in their ideas of the requirements of the situation. The long cycloplegia produced by atropine is quite unnecessary for anyone over fifteen or sixteen years of age—homatropine, properly used, to be followed by eserine, will accomplish the desired result with the loss of the use of the eyes for but one

day, surely not a great length of time to enable one to reach a result, which, if accurate, will ordinarily serve for several years. Of course, the greatly desired drug is one which acts for half an hour after instillation, producing complete cycloplegia for that time and then has its effect vanish, but this seems far away at present.

Some there are who fancy themselves as refractionists who claim their work is quite accurate without a cycloplegic. The following 3 cases are suggested for the consideration of such, each having been carefully refracted, one by each of two ophthalmologists near home and one in a distant city; the ages are given and the lenses worn when first seen, as well as the results under the action of a cycloplegic.

Case I.—Miss H. J., aged nineteen years: O² +0.25 sph. with −0.50 cyl. axis 90 degrees =5 5 ?? With homatropine: O.D. +0.25 sph. +0.25 cyl. ax. 180 degrees =5 4 partly. O.S. +0.50 cyl. ax. 165 degrees =5 4 partly. Not a great difference, but enough to stop her headache and discomfort which she had suffered on near work, indeed she felt more comfortable without the old lenses. Both refractions were done within a year.

Case II.—Mr. G. C. T., aged twenty-one years. Headache and ocular tire. Refracted without cycloplegia three months previous to this examination. Wearing O.D. −1.25 cyl. ax. 180 degrees =5/5 partly. O.S. +0.25 cyl. ax. 90 degrees =5/15. With homatropine: O.D. +0.50 sph. −2.25 cyl. ax. 5 degrees =5 4 partly. O.S. +0.50 sph. −1 cyl. ax. 180 degrees =5 4 partly. Correction of the astigmatism stopped his discomfort.

Case III.—Miss A. G. F., aged forty-two years. Blur and ache upon near work. Wearing glasses prescribed six months previously. O.D. +0.50 cyl. ax. 90 degrees =5/5. O.S. +0.75 cyl. ax. 90 degrees =5 5. With homatropine: O.D. +1.25 sph. +0.25 cyl. ax. 90 degrees =5 4 partly. O.S. +1 sph. +0.50 cyl. ax. 90 degrees =5 4 partly. Prescribed less +0.25 sph. with relief from discomfort.

Anyone can do this: it takes but a portion of a day as far as the patient's time is concerned, and is accurate. Then why not do it? Lenses, especially for the correction of astigmatism, are either correct or incorrect: if the latter, the patient will not be relieved and will waste the time and money seeking for the cause of the

discomfort. It is probable that almost all cases could be refracted satisfactorily without the use of a cycloplegic in the absence of astigmatism, but how are we to be positive as to the presence or absence of astigmatism until we have used a cycloplegic? Many cases will cover up a fairly high astigmatism and suffer as a result, as the following case shows:

Mrs. J. B., aged twenty-two years, had suffered from attacks of trifacial neuralgia which had been diagnosed as tic douloureux and for which an alcohol injection was decided upon, when it was suggested that it might be a reflex from ocular strain and that as yet the eyes had not been examined. The ocular examination showed no change in structure, visual acuity in each eye slightly better than normal, near point consonant with her age and a good muscle balance. It was not believed that the eyes were at fault, but, as a precautionary measure, homatropine was instilled and the surprising amount of one diopter of hyperopic astigmatism was uncovered in each eye, the correction of which relieved the symptoms for over a month, when the patient reported a return of the symptoms. It was noticed that the frames were not those brought for inspection after the refraction, and it was found that she had not liked them and had the lenses placed in different frames by another optician, who had the cylinders about 15 degrees off axis. When this was corrected there was again a disappearance of the discomfort.

Here was a case of ocular strain simulating a serious condition, which at times leads to radical operative procedures, and which probably could not have been accurately measured and corrected by any means other than the use of a cycloplegic. Was not the very short time given to make the examination well spent?

The men who did the work upon which Dr. Mitchell bound his statements were all careful and accurate observers and made sure that their results were correct. In one case, a man fifty-one years of age, atropine was used to determine his error, as it was felt that the accommodation was too active for a proper manifest refraction. They took the trouble to impress upon their patients the need for painstaking care and to secure their coöperation, and, unless miracles are expected, their footsteps may be followed in these days of haste.

Discussion. Dr. Burton Chance said that we should feel much indebted to Dr. Langdon for reviving, in this Section, the subject of a deeper interest in eyestrain, and for his allusions to Weir Mitchell's solution of many of the

complaints depending upon the need for spectacles. Without in any way lessening the respect for Weir Mitchell, he would speak for a fuller recognition of the labors of the early ophthalmologists of this city who founded what has been called the "Philadelphia School of Ophthalmology." It was in the course of the careful scientific study of individual cases, under their care, that Weir Mitchell's attention was called to the value of the correction of refraction defects, and through which he was able to apply their results to the quieting of his nervous patients and to the restoration of the health of many who consulted him. He, in his helpfulness, turned to Dyer, Thomson, Norris, Harlan, and others; it was their patient and prolonged testing of the ocular deficiencies and the adjustment of spectacle lenses that brought about relief. Mitchell's papers served to broadcast the information and practice of these pioneers in refraction at a time when their endeavors and competent observations were denied the credence they so well deserved. It was of interest for him to recount that today, while rearranging on his shelves the personal case-books of Dr. William Thomson, which he possesses, he saw in the records of 1871 the notes of certain baffling cases whose health had been restored by the adjustment of cylindrical glasses of low measures, and of Thomson's early consultations with Weir Mitchell. It was of further interest to observe that most of the refraction records relate to myopes and presbyopes; but toward the end of the book for that year there came the first of interest to us in the aspect we are now considering. It was that of a young lady who regularly passed sleepless nights whenever she attempted to read or perform other close occupations in the evening while her family and their friends were gathered together in the poorly gas-lighted sitting-room of her home, the atmosphere made still more hazy by the thick smoke from the men's tobacco. This lady's relief when she wore cylindrical glasses was indeed remarkable.

Dr. McCluney Radcliffe: I congratulate Dr. Langdon on his timely and practical paper and on his insistance of a careful and methodical refraction under full effect of a mydriatic. I have always been opposed to the so-called quick or "one hour" use of a mydriatic, as I am convinced that an accurate estimation of refractive errors cannot be determined in that manner. While it is possible to paralyze the accommodation in that length of time, it is not possible to rest a tired eye with a retina fatigued and irritated by overuse or by uncorrected or improperly corrected refractive errors. I fear we frequently overlook this important factor in our refraction and it is especially true of low errors in highly sensitive eyes which are the most difficult to test. We are all familiar with the improved vision on the second test where the eyes have had at least twenty-four hours rest under a mydriatic. Recently I had a case which illustrates the necessity of a complete and moderately prolonged mydriatic action. An active business man of thirty-nine years, using his eyes for close work, was wearing: Right, −0.50 cyl. ax. 15 degree; left, −0.37 cyl. ax. 180 degree. He complained of tired eyes and frequent attacks of frontal headache. He was examined by a skilful oculist, but unfortunately he did it in the quick way, only using homatropine for one hour. After I had continued the homatropine for twenty-four hours, my result was: Right, +0.50 cyl. ax. 105 degree; left,

+0.25 cyl. ax. 90 degree. Since wearing that correction his headaches have entirely disappeared and he is using his eyes with comfort. I find that patients are perfectly willing to take the time for thorough refraction when the necessity for it is fully explained to them.

Dr. Edward A. Shumway said he had seen prolonged mydriasis after homatropine had been prescribed, but this had been due to a drug wrongly labeled by the manufacturing chemist, and he thought that in a case cited by Dr. Buchanan this error had occurred, and hyoscyamine or one of its isomeric preparations had been used in place of homatropine.

OCTOBER 21

Exhibition of a Case of Extreme Traumatic Enophthalmos

Dr. William Zentmayer presented L. D., colored, aged sixty-two years, who was injured in an automobile accident December 12, 1925. She received a compound comminuted fracture of the skull involving the orbital bones, with laceration of the soft tissues, which necessitated the enucleation of the right eye. She came to the Wills Hospital on May 21, 1925, to learn whether anything could be done to make the left eye useful.

There is evidence of the injury in the nature of flattening of the bones in and about the orbits, with exostoses and scars in the soft tissues, causing contraction of the palpebral fissures. On the right side the socket is in a fairly healthy condition. In the left orbit there is a mass of granulation tissue just behind the semilunar fold.

There is a high degree of enophthalmos, the globe being fixed against the inner wall of the orbit midway between the base and apex. The eyeball appears to be of normal size. The cornea is hazy, and the anterior chamber apparently of normal depth. There are several posterior synechiæ. The vision is reduced to hand movements.

During the late summer of 1926, she had an abscess of the lacrimal sac on the right side, which was treated by Dr. Tassman. The patient states that the discharge formerly came from the orbit on this side and now has ceased to do so and seems to be coming from the sac.

Examination by Dr. Skillern showed no evidence of ethmoidal disease nor any connection between the ethmoids and the lacrimal fossæ.

Exhibition of a Case of Bilateral Symmetrical Congenital Dislocation of the Lens

Dr. William Zentmayer, in presenting the patient, purposed to show the result secured by the operative procedure employed in the left eye.

The child, who is seven years of age, was shown before the Section last spring, and there was considerable discussion as to the best method of treating these cases.

Four discissions were done on the left eye. The vision, with correcting lenses, is now 6/5 pt. The pupil is active. When dilated, only a trace of capsule can be seen near the pupillary margin down and in.

Two needlings have been performed on the right eye. All the operations were done under local anesthesia.

Hematogenous Siderosis Associated with Angiosclerosis of the Retinal Vessels and a History of Diabetes

Dr. T. B. Holloway presented for examination a woman, aged forty-five years, whom he had examined at the University on the service of Dr. Stengel. Upon admission, she complained of blindness of the right eye and impaired vision of the left. The patient was a housewife, and denied any trauma affecting either eye and any acute inflammation of the eyes. There was a history of diabetes dating back two years, although at the time of admission to the hospital her urine was sugar-free and her fasting blood-sugar 0.143. Her blood-pressure varied from 186 to 112 to 210 to 130.

The right eye was blind; vision of the left, 6/9. The right iris was greenish-brown, the left blue. Examination of the eye with corneal microscope and slitlamp revealed several pinpoint grayish dots on Descemet's membrane; the aqueous was clear. There was a partial absence of the uveal border to the pupil. Scattered over the anterior surface of the lens, and arranged in an irregular circular or wreathlike manner, there was a series of a dozen or more small, circular lesions with central rust-colored spots; these lesions extended through the capsule into the anterior stromal layers. On the capsule, down and in, there was a small nodule of uveal pigment, evidently the site of small synechia that had torn loose.

In the lens there were a number of isolated, dotlike opacities. The extreme posterior cortical layers appeared buff-colored. In a

careful search over this area, a translucent crescentic area could be noted, and, in endeavoring to see the vitreous through this fissure, one could note a wavelike motion at the buff-colored edge, doubtless due to a free margin of pigmented exudative layer covering the posterior surface of the lens. No view could be obtained of the vitreous. With the ophthalmoscope, no fundus reflex.

Left Eye. Peripheral lens striæ with punctate opacities and hemorrhagic masses in the lower portion of the vitreous. The disk was pale, the vessels showed excessive angiosclerotic changes. Below the macula there was a large preretinal hemorrhage. In the macular region there was a cluster of tallowlike exudates such as are seen frequently in so-called diabetic retinitis. There was a large hemorrhagic area in the lower midzone. The lower periphery could not be seen owing to the hemorrhage into the vitreous.

Dr. Holloway referred to the literature and the few cases of this type that come under observation. He tated that it was the first typical case of this character that he had observed.

Presentation of Instruments used by the Late Dr. William F. Norris

Dr. Burton Chance said that to those present who had been associated with Dr. Norris, the sight of these instruments must bring a pang of remembrance of his greatness and his usefulness.

It is fitting that these instruments should be given into the keeping of this Section of the College, because, through Dr. Norris' enthusiasm for the development of ophthalmology in Philadelphia, he became one of the founders of the Section, and in its affairs he manifested the keenest interest to the last of his days.

So pronounced was Dr. Norris' personality, and, through his teaching, so permeating his influence, that, to some who survive him, he seems to be still in the flesh and ever ready and willing to counsel and advise them, as well as to share their enthusiasms for their own new-found facts and methods of treatment. It does not seem possible that he has been gone from us twenty-five years.

William Fisher Norris was born in Philadelphia in 1839, the son of a distinguished Fellow of this College. After graduating from the University Medical School, then on Ninth Street, he entered the Union Army as an assistant surgeon and served throughout the War. While in the Army he became interested in photography,

and from that pastime he passed to the elucidation of questions in optics, and later he desired to study the diseases of the eye. To this end he determined to visit Vienna.

This step was taken in spite of the fixed opposition manifested by his devoted father and his many professional friends, who could not conceive, so he told the writer, that one could confine himself to the care of a special organ of the body from any high and ethical motive. Despite his personal wealth and social position he went, nevertheless, and became the devoted and industrious student of Arlt, Jaeger and other teachers who were creating ophthalmological science as we now know it. His individual researches were remarkable for so inexperienced an American student.

Upon his return to Philadelphia he established a public and private practice which was recognized only after he had overcome the obstinate opposition of his friends and the practitioners whose support he longed to have. Dr. Chance did not enumerate the many positions that Dr. Norris held, but alluded to them only to draw attention to his painstaking devotion and indefatigable zeal in all he undertook. In time, largely at his own expense, an ophthalmological department was established at the University; indeed, the Hospital itself owes much to his initiative and unfailing support.

For many years he was surgeon at the Wills Hospital. It was there that Dr. Chance had the honor to serve him as house surgeon and there shared his friendship.

Dr. Norris, in his numerous writings, was most courageous in publishing the results of his studies, his clinical observations and the effects of his surgical and therapeutical methods; in all he sought to teach as well as to show forth his own successes. The College possesses all his writings, which are sound, and certain have become classics; all are worth reading today. He was modest as to his own powers and commonly diffident in the presence of strangers and before those whom he deemed unenlightened in the subjects about which he intended to speak. To his friends and colleagues he was kind, loyal and unaffected. His contributions to the proceedings of this Section were recalled. At those meetings he assumed that all were cognizant of the principles which he elucidated, in all of which there were charm and directness still unexcelled by his successors.

As an operator he was not brilliant, though he was calm, deliberate and painstaking. He never operated for the sake of operation.

With the sick poor, in his hospital practice, no one was so gentle, so patient, so considerate as was he; but woe to the man or woman, on the pretense of worthiness obtaining admission to the wards, who tried to impose upon his generosity. Dr. Norris' ideals governing the conduct of the medical life were of the highest and noblest, and he labored to advance ophthalmology to a fit place in the sciences; with what success he labored the endurance of this Section well attests.

Dr. Chance said: "There are some here who knew him more intimately, perhaps, and for more years than did I, yet they cannot have had a greater respect for him than was mine, and, while he, years before, might have been considered the Father of Ophthalmology in his community, he was indeed as it were, my 'father' in the study. It is with that affection that I offer, in the name of Dr. George Norris, these cases of instruments to be preserved in the College in memory of one of its most distinguished Fellows."

Vaccination Ulcer of the Eyelid

Dr. Burton Chance wished to have recorded a case of vaccine ulcer of the eyelid, as it is but the fourth case he can recall having seen. He regretted the patient had not put in an appearance. She is a Southern negress who had never been vaccinated, but her young girl was, recently at school. Three or four days after which the woman's left upper lid began to swell, and a small pimple developed. About ten days ago she applied at Wills Hospital in the morning hours for relief of the swollen and painful lid. The house surgeon lanced the lid, naturally seeking to relieve the tense lid, but there was no effusion. In the afternoon we observed a more or less round vesicular papule on the tarsal border, which in a day or two became crusty and assumed the aspect of a vaccination sore as seen elsewhere. It has progressed as would an ordinary vaccination lesion.

Juvenile Cataract, Secondary to Uveal Disease

Dr. C. R. Heed exhibited a girl, aged four years, with cataract of the right eye, secondary to uveal disease. The child had been in perfect health, and at no time was there any congestion of the eyes. Early in April last the mother noticed a white film in the pupil of the right eye. Examination at that time showed light projection

of the eye apparently normal; pupil 4 mm., no reaction to light; ocular rotations unimpaired; convergence about 25 degrees. Atropine instilled, and after one hour failed to show any dilatation. The pupil of the left eye was 4.5 mm. and did not react to light.

After the use of atropine for two weeks, there was shown almost complete synechia of the right eye and three-fourths of the pupillary margin of the left eye adherent. Tension normal. The left eye showed a slight wrinkling of the anterior lens capsule, the vitreous clear, disk and retinal vessels healthy. No evidence of retinal or choroidal disease was present. Physical examination and *x*-rays of the chest were normal; Wassermann 1+. Diseased tonsils and adenoids were removed. The atropine was continued, with gray powder internally. In October the ocular condition remained unchanged with the exception of a reduced tension of the right eye. The iris did not push forward, to indicate lack of drainage into the anterior chamber. The case possessed interest from the existence of a prolonged iridocyclitis, with no noticeable signs of external congestion.

Calcareous Film of the Cornea

Dr. Leighton F. Appleman presented a man, M. S., aged sixty-eight years, a hatter by trade, who showed, originally, bilateral calcareous film of the cornea. At this time the film was still present on the right cornea, while the left eye shows the condition following its removal.

This patient came under observation with the statement that both eyes had been affected since 1908, beginning as a film over the sight, which gradually grew more dense. As direct vision became impossible, an optical iridectomy had been done in each eye about 1910, at the lower part of the iris, which had enabled him to continue at his work until he presented himself at the Wills Hospital in September, 1925, on account of failing vision. This failing vision was seen to be due to cataract in each eye in addition to the film. It was decided to remove the cataracts, which was done without unusual reaction in either eye, and with very little secondary capsular remains.

Vision, following the cataract operations, while greatly improved for objects in his lower field, was still impossible at distance because of the corneal films. These opacities occupied the center of each cornea, corresponding in shape and area to that exposed when the lids were partially closed, being about 5 to 6 mm. wide over the

center and extending laterally almost to the limbus on either side. The epithelium over this area was perfectly smooth, not elevated above nor depressed below that of the clear cornea around this area. In macroscopic appearance it was uniformly white, dense, somewhat granular and superficially located.

With the slitlamp it was of a mosaic character, having the general appearance of hexagonal figures of a grayish granular character outlined by lines of pure white. In one or two places fissures could be seen, running vertically, and in these fissures the substance of the cornea was apparently perfectly clear. It could readily be seen that the condition was very superficial, just beneath the epithelium, and it was decided to attempt removal.

Under local anesthesia the epithelium of the cornea of the left eye was disintegrated over the nasal portion of the opacity by the use of Shahan's thermophore at a temperature of 170° for about forty-five seconds, then, with a small sharp curette, the plaque, which to the touch felt distinctly calcareous, was peeled off in small flakes, leaving the cornea somewhat roughened but otherwise apparently clear.

As it was feared that undue reaction might follow the procedure, only the nasal half of the opacity was removed. However, scarcely any reaction followed and the epithelium had regenerated to such an extent at the end of twenty-four hours that only two pinpoint stains were releaved after the use of fluorescein, although the cornea appeared roughened. This smoothed out within the next two or three days until the surface was level with the rest of the cornea. About one week later the remainder of the opaque area was treated in the same way, again without reaction.

Following removal of the superficial plaque it was seen that a faint haziness of the substantia propria existed which could not previously be recognized. This, no doubt, was due to nutritional changes which allowed the condition to develop simultaneously with the deposition of calcareous material in the superficial structure.

Direct vision in the operated eye was 5/60 a short time after his operation, with the correcting lens which he had previously worn.

Corneal Tumor

Dr. Appleman presented a man, aged forty-eight years, who showed a growth of the cornea of the left eye of about two years' duration, arising just within the upper limbus, firmly adherent,

of a mottled pink color and soft consistency. By pressure of the lid it had become flattened and spread out like a mushroom over the conjunctiva, but not attached thereto as could be demonstrated by the passage of a spatula beneath it. Extension downward as a thin vascular veil or film over the cornea had occurred almost to the pupillary border, so that the vision of this eye had begun to blur.

The patient experienced no pain. As he had just come under observation, no microscopic study had been made, although the possibility of sarcoma of the cornea had been seriously considered.

A New Tucking Operation for the Correction of Strabismus

Dr. Thomas J. Clemens described an operation for correcting squint for which several distinct advantages are claimed over the usual methods now in use. The operation consists of tucking, or folding, the tendon of the muscle upon itself and attaching the distal end of the tuck to the point of the insertion, the folded tuck being depressed between the sclera and the muscle itself. There are only two sutures used for the entire operation.

The patient is placed under a general anesthetic, and the technic is as follows:

An initial incision is made vertically through the conjunctiva about 6 mm. from the limbus, Tenon's capsule is opened and the tendon of the muscle exposed in the usual manner. The tendon is picked up with a strabismus hook and cleared of adhering tissue to the extent of 10 to 12 mm. The tendon is then scarified along the superior fibers to the extent of the proposed tuck.

Two double-armed sutures of No. 6 twisted black silk, 10 or 12 inches in length, are required. The strabismus hook being elevated, one needle of a double-armed suture pierces the tendon from below close to its scleral insertion—the needle being carried through the insertion, picking up some fibers of episcleral tissue in its passage, and emerging through the anterior aspect of the insertion. The needle is then continued through the conjunctiva close to the sclerocorneal junction. This suture may be designated as No. 1 suture. Another suture is passed exactly in the same manner from the other side of the muscle and designated as No. 2 suture. The needles are then removed from that end of the sutures.

The armed end of suture No. 1 enters the posterior aspect of the tendon from below, choosing a point farther back as an angle of the

strabismus is larger, and then passing the needle under the posterior flap of the conjunctiva it emerges on its outer surface, when the needle is removed.

The second suture is passed through from the outer side of the tendon in like manner. A specially designed tucking instrument is now adjusted as follows:

The female member is passed beneath the tendon while the male member is passed over the tendon and beneath the sutures. The tucker is now adjusted to the amount of correction desired, forcing the loop of tendon into the space between the sclera and muscle. The sutures are then tied, the assistant tying one and the operator the other, and the wound drawn together fairly firm, bringing the wound into apposition, while the tucker is still *in situ*. The tension is then relieved from the tucker and the arms are allowed to come to an even plane, when it is withdrawn. The sutures are then tied securely, closing the wound, and at the same time bringing the fibers of the tendon into apposition.

There are several distinct advantages gained by this operation: (1) There is no destruction of tissue; (2) Ease in operating; (3) anterior fibers of tendon brought forward and united at their original insertion—a real advancement; (4) ability to control the amount of tuck; (5) suppression of tucked loop, leaving no unsightly lump; (6) very little reaction and practically no scar.

Discussion. Dr. B. F. Baer, Jr., stated that he had the opportunity of seeing a certain number of these cases upon whom Dr. Clemens had performed his modified Todd tucking operation. In each of these cases Dr. Clemens had performed, in addition, a tenotomy upon an internal rectus muscle. The results were excellent and there was not the lumping of the tucked muscle which is the objectionable feature in the Todd operation. If he were to do this operation, he would not combine it with an immediate tenotomy of an antagonistic muscle, but would defer the tentomy until a later date.

Dr. Edwin B. Miller reported that he had performed a tucking according to the method of Dr. Clemens and had found it all that had been claimed for it.

Instead of ether anesthesia, he had used novocaine and cocaine, injecting the novocaine well back over the muscle to be operated upon. Doing this operation for the first time, he did not get the stitches back far enough and, as the eyes were not quite in parallel, he did a graduated tenotomy on the opposite muscle. Dr. Miller stated that he had the opportunity of seeing some of the cases operated upon by Dr. Clemens two or three weeks following the operation and the results were almost perfect. No reaction, no swelling or lump over the muscle, and full movement in all directions.

Dr. Luther C. Peter stated that he believed that there is a distinct field for tucking in the lower degrees of squint and the higher degrees of heterophoria. The difficulty which he has experienced in the past in tucking operations is the tumefaction which occasionally appears after the use of chromasized catgut. In a number of cases this tumefaction will last for a couple of months. They are rather distressing to the patient and annoying to the surgeon, but ultimately become absorbed and leave no scar. In a few instances he has been obliged to remove the catgut which failed to absorb. More recently, however, he has found plain catgut quite satisfactory, and has not observed such swelling over the site of the operation. The distinct advantage, however, which he sees in the method of introducing the sutures, as proposed by Dr. Clemens, is the use of silk instead of catgut. If our technic can be changed so as to use silk instead of catgut, he believes there would be a distinct advantage gained for this operation.

Dr. Burton Chance stated that he had witnessed a number of Dr. Clemens' operations, from the second of his cases. He was impressed by the accuracy of the adjustment of the parts, and he noticed that the reaction and infiltration of the conjunctivoscleral tissues was not as great as he had observed in other forms of tucking operation.

Corneal Lesions

Dr. G. E. de Schweinitz and Dr. Perce De Long exhibited a series of microscopic slides indicating various lesions of the cornea, associated with the exhibition of a number of lantern slides illustrating slitlamp appearances of similar corneal diseases.

NOVEMBER 15

Exhibition of a Case of Sarcoma of the Iris

Dr. William Zentmayer presented a case of tumor of the iris. The patient was a boy, aged ten years, referred to me at the Wills Hospital by Dr. Calvin Rush, November 1, 1926, with a history that for the previous four months a small, pinkish growth had been noticed over the color part of the left eye, which had been slowly increasing in size. There was no history of injury or recent inflammation.

In the left eye there is a flesh-colored tumor about $3\frac{1}{2}$ x 4 mm. in size. It is smooth and ovoid, and rests on the surface of the iris at the small circle and projects into the pupillary space. Fine bloodvessels are seen ramifying on the anterior surface of the growth. The iris reacts to light except at the part on which the growth rests. The vision is 5/5.

With the slitlamp the growth is somewhat translucent but not cystic. The surface is covered with ramifying vessels coming from the surface of the iris. The vessels on the iris tissue are much larger and more tortuous than those on the growth proper. There is an irregular attachment of the tumor to the iris by means of several strands of pale, pinkish-white tissue, which is also vascular. No precipitates are seen either on Descemet's membrane or on the anterior capsule of the lens. The diagnosis is probable leukosarcoma of the iris. These growths appear as early as the second and as late as the seventy-fifth year. They are, as a rule, pigmented, of slow growth and not accompanied by inflammation until a late stage. There may be hemorrhage into the anterior chamber. The diagnosis rests upon the noninflammatory, slow growth, the flesh color, the vascularity and rounded form, and the position on the iris at the smaller circle.

Gumma is usually accompanied by iritis, other syphilitic symptoms, is rust-colored or yellowish-white, there is frequently hypopion, and the growth is often peripherally situated. Tubercle is generally accompanied by mild inflammatory symptoms, is yellowish-white or grayish-white in color, is of rapid growth, and located most frequently on the smaller circle. There is hypopion occasionally.

There are probably less than 100 cases of sarcoma of the iris on record. Ball states that cures have been obtained by iridectomy and removal of the mass by Veasey, Arlt, Kipp, Knapp and others. Casey Wood, Brown Pusey and Ball believe that enucleation should be done as soon as the diagnosis is made.

In this case it is proposed to attempt the removal of the tumor by iridectomy, using either the keratome and enlarging the incision by the scissors, or by using the Graefe knife, taking care in either case not to cut the growth with the instrument for fear of disseminating the tumor cells.

Dr. Zentmayer expressed his indebtedness to Dr. J. S. Shipman, of the resident staff of the Wills Hospital for the excellent color sketches which he had made.

Traumatic Enophthalmos

Dr. C. R. Heed exhibited a man, aged twenty-seven years, who entered the Jefferson Hospital with enophthalmos which followed a direct concussion of the right eye by a stream of water from a fire hose. While there was edema and discoloration of the lids, a sub-

conjunctival hemorrhage and a small laceration of the conjunctiva, there was no evidence of a rupture of the globe. The cornea was clear, the anterior chamber of normal depth, the tension normal and the pupil round, 5 mm., and did not react to direct light. There was marked recession of the eyeball, the Hertel instrument recording 7 mm. and the left eye 16 mm. Upon discharge from the hospital, ten days after the injury, the vision of the right eye was 6/7.5; left eye, 6/5; rotations showed marked impairment downward, inward and outward, and no perceptible movement upward. The eye-grounds were normal.

Dr. Heed said that the mechanism of enophthalmos following penetration wounds was different from those where the condition followed severe blows on or in the region of the eye, with no apparent evidence of orbital fracture or signs of inflammation. The explanation of the latter cases has been variously stated as due to vasomotor disturbance, trophic changes affecting Tenon's capsule or Mueller's muscle, atrophy of the orbital fat, or palsy of the sympathetic nerves.

The supporting structures holding the globe in position have been given as trabeculæ from the periosteum to Tenon's capsule and the check ligaments. No prominent mention has been given to the oblique muscles with their aponeurosis as factors in this connection, yet one must concede such a function when considering the course of these muscles. Both are held anteriorly to the orbital rim and pass backward to be inserted into the sclera back of the equator.

The initial causation of the enophthalmos in this case has been assumed to be a rupture of the supporting trabeculæ and a probable separation of the tendon of the inferior oblique, with vasomotor disturbances which resulted in evacuation of the orbital vessels.

Sarcoid of the Eyelids

Dr. Louis Lehrfeld stated that at the request of his associate, Dr. Mitchell Bernstein, he had examined the eyes of Mr. S. M., aged fifty years, August 19, 1926, having been referred because of ptosis of the right upper eyelid. Examination revealed two nodules in the body of the lid and one on the border at the outer third. These nodules resembled chalazia except that the skin covering them had a blusin tinge, and that at the lid border resembled a nevus. The eyelid was quite stiff, resisting eversion, which was

accomplished only by considerable effort. The mucous membrane beneath the site of the nodules was hyperemic, very much like that accompanying larger chalazia. Similar nodules, firmer in appearance were noted on the lobes of the ears. Flat, slightly elevated and broader skin lesions were found on the chest, back, thighs, abdomen and fingers. These were a deep red-wine color, well circumscribed, round, oval or quadrate. The entire skin of the body had a cyanotic appearance.

The eyelid lesions were part of a general skin disease. The patient presented the following constitutional symptoms: Cough, hemoptysis, cardiorenal disease, edema of the lower extremities, a polycythemia followed, later, by secondary anemia and moderate leukocytosis.

When seen again, September 23, the two nodules in the body of the right eyelid had extended in size, occupying nearly the entire surface of the skin, and by mere weight and mechanical obstruction prevented an eversion of the lid and producing a ptosis of such degree that the palpebral fissure was almost closed. Another violaceous nodule had appeared near the inner canthus of the upper lid since the last examination, four weeks previously. There had also appeared, for the first time, a nodule on the left lower lid near the external canthus, and another high up in the lid just below the eyebrow. The fundus showed no disease and the refractive error was two diopters of myopia in each eye.

Since September 23 the patient experienced a change in the character of the nodules, the large elevated lesions being softer and dark red, others lighter red. Those on the eyelids appeared to become more extensive, with other flat patches over the eyebrows of both eyes.

The case was referred to Dr. Mitchell Sidlick, a dermatologist, who diagnosed the case clinically as one of Beck's sarcoid, and later verified the diagnosis by pathologic examination of an excised nodule taken from the skin of the abdomen. Pathologically the lesions resembled very much that of tubercles in tuberculosis.

Dr. Sidlick reported the histological examination, as follows: "Cell nests resembling tubercle formations are found, separated from each other by connective-tissue substance. A cellular infiltration in the tubercles is composed of epitheliod connective-tissue cells. The corium shows a connective-tissue cell proliferation. In some places capillary loops of papillæ are present."

Dr. F. H. Verhoeff describes the microscopic examination of Derby's case, reported in 1917, as follows: "There is seen a conglomeration of tubercles, partly or wholly separated from each other by septa of connective tissue. All of the tubercles seem to be situated in distended lymph spaces, the involvement extending through the entire thickness of the skin into the underlying fat tissue and muscle. The tubercles are always separated from the surface epithelium by a layer of connective tissue. Under low power the general configuration closely simulates the appearance presented by an epithelioma of the skin which is invading the lymph spaces. The tubercles vary greatly in size, the largest occurring in the superficial lymphatics and the smallest in the deepest layers and at the periphery, where the process is evidently extending. The tubercles are made up of typical epithelioid (endothelial) cells, which have gone on to the abundant formation of giant-cells, many of the latter being of the Langhans type. The larger the tubercles the larger and more numerous are the giant-cells. Bloodvessels are only occasionally seen within the tubercles. The tubercles show no infiltration with pus-cells, but there is slight infiltration with lymphoid cells, generally at their peripheries. There is no necrosis of any kind to be seen. The stroma between the tubercles is notably free from inflammatory reaction. Sections stained for tubercle bacilli fail to show any organism present." No tubercle bacilli were found in the sections of the case here presented or in the fluid obtained in the tappings for the relief of hydrothorax. The etiology of sarcoid is not known. The case is presented because of the rarity of the disease, especially as to the involvement of the ocular structures. Derby reported a case in 1917 as the first in ophthalmic literature and Dr. Lehrfeld wished to add this one to the records.

Discussion. Dr. S. S. Greenbaum pointed out that the term sarcoid was adopted originally by Kaposi, and is used to designate lesions resembling sarcomata clinically; but they do not recur *in situ* when removed as does sarcoma, nor do they ulcerate or metastasize. The lesions occur in the form of tubercles, nodes, and are usually multiple and painless. They may disappear spontaneously or following the use of arsenic, mercury, tuberculin, etc. Their histologic structure is often tuberculoid. Several clinical types are described: (1) Cutaneous sarcoids or lupoids of Beck. (2) Hypodermic sarcoids of Darier-Roussy. (3) Disseminated nodular or dermic sarcoids. The exact nature of sarcoids is not known: In some it is tuberculosis; in others syphilis, and in others a different infection.

Dr. Edward A. Shumway said that in the case reported by Derby and Verhoeff the growth on the eyelid had been excised and had recurred. The condition is very uncommon, so that oculists have very little experience with it clinically. According to Hartzell, tuberculosis is present in about one-third of the cases, and the fact that it is improved by the administration of arsenic does not, of course, prove that it is syphilitic in origin, as many skin lesions are greatly improved by its use.

In the differentiation of sarcoid from lepra, syphilis or the nodular form of erythematous lupus, with which it might be confused, Hartzell says that the absence of ulceration and the presence of fine yellowish points in the sarcoid were important. The latter could be best brought out by pressure with a glass slide.

Epithelioma of Eyelid in a Child Aged Four Years

Dr. S. S. Greenbaum presented the report of a patient, a child, aged four years, the son of people who were first cousins. The parents first noted, during the first year of life, an increasing freckled appearance of the facial skin. Gradually there developed here and there upon the skin hard concretions which frequently dropped off spontaneously but just as frequently recurred. About six months before he saw the child, one of these hard masses on the lower eyelid became ulcerated; the ulceration failed to heal and progressively enlarged. Histological examination showed a squamous-cell cancer. Diagnosis was xeroderma pigmentosa with malignancy.

About 110 cases of this condition have been reported. It usually begins during the first or second year of life, almost always during the summer and sometimes very shortly after a single exposure to direct strong sunlight. There is, first, a dryness of the skin, with progressive development of freckle-like lesions, which, however, are much darker than ordinary freckles. The exposed surfaces are the parts mainly involved. Slowly there develop small, white, atrophic areas on the skin, with telangiectasis and keratoses. These keratoses usually, after a time, undergo malignant degeneration. The following eye disturbances are found in this condition: Ectropion, due to the atrophic lesions on the eyelids; photophobia, with lacrimation; interstitial keratitis; corneal nodules and opacities.

The exact cause of xeroderma pigmentosum is not known, but it is known to be common in children born of a consanguineous marriage and it is likewise thought that there is a congenital hypersusceptibility to the actinic rays of the sun.

The patient was presented before the Section because of the presence of the squamous-cell cancer of the eyelid and to invite discussion on the etiology of the photophobia. Photodynamic sensitization, or light sensitization, has been known for years. Recent research appears to indicate that there are at least two photosensitizing substances in the cells or the circulating blood of the capillaries responsible for the clinical manifestations. According to Harris and Hoyt, it is the amino-acids of the proteins which act as the sensitizers. It is not at all unlikely that this latter, when present in excessive amount in the skin, is likewise present in excessive amount in the exposed surfaces of the eye, that is, in the conjunctival capillaries and the lymph spaces of the cornea. It is interesting to note that the photophobia was made distinctly worse following the local injection of mercurochrome. The explanation for this would appear to be that mercurochrome is a compound containing fluorescein, which itself is photodynamic.

An Unusual Case of Medullated Nerve Fibers

Dr. A. G. Fewell exhibited a case of medullated nerve fibers which was seen in the eye clinic of the University of Pennsylvania. The patient was a colored boy, aged twenty years. There were no other congenital defects present than the one here reported.

The case was shown because of the unusual situation of the medullated fibers in the left eye. It is an excellent example of the arcuate distribution of the nerve fibers on the temporal side of the retina. The fibers are nonmedullated as they leave the disk, becoming medullated a short distance up and to the temporal side. They then pass out toward the temporal periphery, arching above the macula. They lose their medullation in places as they spread out, but can be traced outward and in part end on the median raphé. This area is interpreted in the field by a comet-shaped relative scotoma which runs into a cut in the lower nasal quadrant. The blind spot is slightly enlarged laterally. The vision with correction is O.D., 6/6; O. S., 6/6?

Discussion. Dr. Luther C. Peter said that Dr. Fewell's case presented a very interesting picture. He was much interested to know whether Dr. Fewell had been able to find any areas of absolute scotomata in the denser opaque nerve fibers. Up to the present time the scotoma found has been relative in type. It is probable that if the form-test object were reduced to standard—a minute angle of probably ten minutes—absolute areas might be detected in parts of the distribution of these nerve fibers.

Dr. William Zentmayer said that the question of the scotoma in connection with the medullated nerve fibers had been thoroughly discussed by Gradle a few years ago. He found that there was no close agreement between the ophthalmoscopic and the field findings. He therefore considered the term opaque fibers a misnomer. It would seem to Dr. Zentmayer that this term was applied to them from the viewpoint of the observer and not the patient. They certainly appear opaque. A point brought forward by von Hippel and Openheim, that they occur relatively frequently along with other stigmas of degeneration in physically abnormal people, he had been unable to confirm.

Observations on the Surgical Treatment of Senile Cataract Complicated by Glaucoma

Dr. Luther C. Peter submitted a series of 26 representative cases for discussion. In the first group of 10 cases, 5 had hypermature cataract complicated by glaucoma, and 5 were mature. Five extractions in capsule yielded 2 good results, 2 fair or surgically satisfactory, and 1 poor. Five extractions by capsulotomy yielded 1 good result, 1 fair, and 3 unsatisfactory. In the total of 10 cases, all recorded as poor were cases of primary glaucoma. Out of 3 of these the glaucoma was subsequently controlled in 2 instances by cyclodialysis, and in 1 by sclerocorneal trephining. The final results were 5 good, 4 fair and 1 poor.

In the second series of incipient cataract with primary glaucoma, 11 cases were trephined, with 11 good results. Three were treated by cyclodialysis, with good results in 2, and fair in 1. One iridectomy yielded poor results. The sixteenth case was one of complicated cataract and secondary glaucoma following uveitis, in which an iridectomy arrested the process.

One of the points of special interest is the frequency of this dual condition in the same eye. This is to be expected in primary glaucoma because of the chronicity of the disease accompanied by lowered ocular nutrition and probably, in most cases, by a subsequent low-grade uveal disease. In mature and hypermature cataract the incidence of secondary glaucoma is conditioned upon increasing lens toxicity and mechanical blocking of the angle. The latter factor probably is the most important.

General Management. As a prophylactic measure it is advisable to operate upon a mature cataract even though vision in the fellow-eye is good. It is the logical and only safe measure to prevent the development of secondary glaucoma.

Primary glaucoma complicated by incipient cataract is best controlled by sclerocorneal trephining. Iridectomies are of less value, first, because they fail too often in uncomplicated cases of glaucoma; second, the added danger of blocking of the angle by the swelling lens almost surely demands a third operation, after the extraction of the lens, to insure the control of the glaucoma; third, after the lens is extracted, the upper angle is apt to be blocked by capsule or iris tags, inflammatory or lens débris. If the cataract is well advanced and extraction near at hand, an iridectomy may be practised with the expectation of a cyclodialysis or trephining after the lens is extracted.

Mature and hypermature cataracts should be extracted in capsule when possible. Extraction by capsulotomy is less satisfactory. The technic recommended for intracapsular extraction is by Kalt forceps combined with gentle pressure over the lower part of the cornea by a Smith hook. Extraction in capsule is more apt to arrest secondary than primary glaucoma.

Residual glaucoma, or glaucoma after extraction of the lens, if the hyaloid is intact, may be arrested by trephining or by cyclodialysis. The trephine opening should not be placed in the area of the iris coloboma, nor yet in the neighborhood of the cataract incision, but in the lower limbus, or where the anterior chamber is deepest. Cyclodialysis has yielded satisfactory results in 3 of the author's cases.

Discussion. Dr. P. N. K. Schwenk expressed his appreciation of Dr. Peter's paper, but asked if Dr. Peter had ever tried the triple operation of, first, posterior sclerotomy; second, iridectomy; and third, extraction of cataract at one and the same time?

He cited a case that came to him at Wills Hospital about ten years ago, in which a colored woman came to him with inflammatory glaucoma, with no light perception, suffering intense pain. In consultation with his then colleague, Dr. Wisher, it was decided to admit the patient to the hospital for an iridectomy; but, instead, he did a posterior sclerotomy, irridectomy and lens extraction, all at the same time. This was done under general anesthesia. A roller bandage was applied, with instructions not to open the eye for three days. She made an uneventful recovery. She came back in six months with the eye quiet, and, to his surprise, had 20/30 vision. Later, he did several others, but without visual results. This gives a complete relief of tension.

In replying to Dr. Schwenk, Dr. Peter stated that he had not practised Dr. Schwenk's procedure in exactly the same manner as he had suggested. It is, however, a logical procedure and would undoubtedly render the iridectomy and extraction of the lens safer than without this measure.

The question of cyclodialysis is as yet *sub judice*. In clinics, such as those in Vienna, where the procedure is largely practised and well thought of, the percentage of successes claimed for the operation is rather low. It has seemed to him, however, that we have not selected our patients with sufficient care for the application of this operation. The lowered tension which follows a cyclodialysis is not due to suprachoroidal drainage, but to the opening up of the canal of Schlemm. The suprachoroidal space is walled-off quite early. It would be impossible, therefore, to expect a cyclodialysis to be of any great value in inflammatory cases—cases in which the canal of Schlemm is closed as the result of inflammatory process. It is more likely that the operation will succeed in the noninflammatory types, in which the blocking is more or less of a mechanical character. It is quite probable that if greater care is exercised in the future in the proper selection of the cases for cyclodialysis the value of this surgical procedure may receive a better rating.

Ocular Fields in Syphilis: Retinochoroiditis in Early Tabes

Dr. William F. Bonner (by invitation) discussed ocular-field changes in syphilis and exhibited a patient showing retinochoroiditis in early tabes: (1) *a*, The annular scotomas show the amount of intraocular disturbance; *b*, the fields for form and color suggest the intraocular lesion. (2) Neurological examination shows early tabes. (3) The last examination of the fields showed increase of intraocular and intracranial conditions.

Discussion. Dr. William Zentmayer said that the most striking feature in the fundi of Dr. Bonner's patient is the sclerosis of the choroidal circulation. The areas of choroiditis are inconspicuous and there are numerous cholesterin crystals scattered about the fundus. It is probable that the choroidal sclerosis is the essential feature and better explains the ring scotoma than do the areas of choroidal atrophy. He stated that he was not taking exception to Dr. Bonner's diagnosis, as this seemed unfair when he has had the opportunity to study the case thoroughly, whereas we have had an opportunity for a hasty glance.

Dr. Luther C. Peter said that the presence of interlacing of color fields in clinical work indicates one of three things: First, it may be an artefact; second, it may be indicative of hysteria; and third, it may be indicative of choroidal disturbance. If the interlacing is between red and green, the findings should usually be regarded as artefacts or as evidence of hysteria. If the interlacing is between red and blue, the possibility of choroidal disturbance should receive very careful consideration. Dr. Peter said he did not have the privilege of seeing either Dr. Bonner's patient or the fields, but it was probable that interlacing of the color fields, of which he speaks, may be evidence of choroidal involvement to which Dr. Zentmayer has referred.

DECEMBER 16

A Further Report on a Case of Primary Sarcoma of the Iris

Dr. William Zentmayer presented the microscopic specimen in the case of tumor of the iris shown at the November meeting. The growth had been removed by iridectomy. A keratome was introduced at a sufficient distance from the position of the growth so as to get as large a limbal incision as possible without the knife coming in contact with the growth. The wound was then enlarged with the scissors. The iris was seized between the base of the growth and the ciliary attachment and withdrawn from the anterior chamber. A minute fragment of the tumor was scraped off and was seen resting on the posterior surface of the cornea near the wound. Free bleeding prevented attempt at removal. Forty-eight hours later, the eye being quiet and the anterior chamber restored the wound was reopened with the iris repositor and the fragment removed with the lens spoon.

The eye has remained perfectly quiet and the boy is receiving *x*-ray treatment from Dr. Pfahler.

A Case of Embolism of a Branch of the Central Artery of the Retina

Dr. George H. Cross presented Miss M. W., white, single, aged twenty years, who first consulted him about two weeks previously because of inability to see, with the right eye, objects below a central horizontal line. This condition was first noticed following what she termed "a bad spell with her heart" about one month previously. Her past medical history shows that she had influenza in 1919, scarlet fever five years ago, and numerous spells with her heart.

Ophthalmoscopic Examination. Media clear, about one-half disk diameter from the upper temporal aspect of the disk the superior temporal artery seemed to stop suddenly, beyond this there was a very thin, threadlike stream of blood in the center of the artery, the walls of which seemed to maintain their original diameter. This fine stream and the vessel walls were lost in a large area of "ischemic necrosis," irregular in outline, about the size of the disk, and then emerged on the opposite side as a broken column (streaming) for a short distance, then as a vessel of full size. Just before

the embolism was reached in following the course of the artery from the disk, from a small branch artery there was observed a fan-shaped group of vessels (arteries of new formation) which extended into the ischemic area. The greenish color in the center of the ischemic, necrotic area was diagnostic of an infective embolus from a heart-valve leaflet. The visual field chart shows complete obliteration of the lower field from a line just below the horizontal and extending downward.

Discussion. Dr. William Zentmayer said that from the statement of Dr. Cross that the opacity in the retina at first had a greenish cast, with the fact that there is localized visibility of the walls of the vessel at the position of the obstruction, it occurred to him that the embolism may have been of a mild septic nature.

An Interesting Condition of the Cornea

Dr. Burton Chance and Dr. Louis Lehrfeld (by invitation) presented from the clinic at Wills Hospital a case of disciform keratitis of the right eye of a robust girl, who had been under observation since later in September. The first symptoms complained of were those of mild inflammation, redness and slight pain for the previous six months. The flow of tears was out of all proportion to the amount of the other symptoms.

When examined, there were supracorneal injection and a whitish disk at the summit of the cornea, slightly obscuring the pupil. The disk extended to within 3 mm. of the limbus, the intervening circle being quite clear.

The corneal disk itself was defined by an outer, dense-white circle of infiltration, and an inner plaque, connected with each other by fine radiations across the translucent intervening space. This anomaly was situated beneath the epithelium, and continued with no visible signs of vascular communications with the limbus until the last week in November, when the slitlamp microscope revealed tiny bloodvessels at the lower extremity of the vertical meridian and the outer of the horizontal.

Dionin and holocain, each in 1 per cent solution, were used. The inflammatory signs gradually disappeared and the eye became quiet, only a shallow whitish ring of about 4 mm. in diameter persisting. The plaque became less dense, the ring appeared whiter. Later on, the outlines became less distinct and irregular, and, as now seen, the infiltration is fading.

No cause for the disease could be ascertained; the blood reactions were negative.

The case was presented because of the tender age of the patient, besides being of sufficient rarity to merit exhibition.

The Crutch Spectacle as an Aid in the Study of Ptosis Associated with Paralysis of the Globe Muscles

Dr. Thomas B. Holloway exhibited a young girl who came to the University Hospital on account of a complete ptosis of the right lid. Upon examination, a complete superior rectus palsy also was found. The eye was amblyopic. No fundus lesions were present. An operation for the ptosis was especially desired, but, with the lid elevated, only 1.5 mm. of the upper portion of the cornea could be seen, owing to an associated spasm of the inferior rectus.

It was decided to do an advancement of the superior rectus as an initial step, and, considering the marked amglyopia, a tenotomy of the inferior rectus. An astonishingly good result was obtained, although, as would be expected, not absolute parallelism. Before deciding definitely upon the ptosis operation, it was thought that the crutch spectacle, as advocated by Mackay, would be of service in determining the amount of annoyance that might result from diplopia with the lid elevated.

An excellent piece of mechanical work was done by the optician, and the cosmetic result with the glasses properly adjusted is unusually good. They have been worn with but little or no discomfort or ocular irritation. The cosmetic result is really so good that one would hesitate at this time to attempt to compete with it by operative procedures.

Discussion. Dr. Luther C. Peter stated that in addition to the brilliant surgical results which Dr. Holloway obtained in his interesting case, he desired to call the attention of the members to a phase of diplopia which is well illustrated by this patient. Diplopia following a paralytic squint in the young is apt to disappear. Young individuals learn rather readily how to suppress the false image, whereas diplopia in adults is apt to continue as an annoying symptom for long periods, sometimes throughout life.

A patient whom he had been observing during the past year, a lad, aged fifteen, who had suffered a paralysis of the left superior rectus and left inferior rectus as the result of a fist blow, was a good example of the same phenomenon. Although diplopia was marked at first, the boy is not annoyed in the least by double vision, and is even able to catch behind the plate in baseball without a mask—a good test of the absence of diplopia.

Chorioretinitis Juxta Papillaris

Dr. Francis Heed Adler called attention to the fact that in all of the cases reported by Jensen, in 1908, the lesion could be observed ophthalmologically at the edge of the disk, and the visual field defect was always sector-shaped, absolute scotoma extending from the blind spot to the extreme periphery.

Numerous cases have been reported under the heading "retinochoroiditis juxta papillaris, Edmund Jensen type," but by no means all of the cases thus reported show the exact ophthalmoscopic picture described by Jensen, nor do the fields faithfully reproduce the defect which he described as typical.

Hepburn, Roenne and Friedenwald have all questioned the advisability of regarding this as a separate clinical entity apart from other forms of acute, circumscribed, exudative chorioretinitis.

A statistical review of the cases reported in the literature shows that these cases do not differ in etiology or in prognosis from other types of retinochoroiditis. One feature alone, however, suggests a difference in development of this type of chorioretinitis, namely, the field defect.

As pointed out by Peterson, the field defect suggests that the nerve fibers are involved, and Van der Hoeve has proposed the term neurofibrillitis because of this defect. In this respect the disease differs from other types of chorioretinitis in which the field defect suggests involvement of the deeper layers of the retina.

Discussion. Dr. William Zentmayer asked how many of the cases of typical Jensen's type of choroiditis in the literature had precipitates on the posterior surface of the cornea and membranous opacities in the vitreous? It would have been of further interest had Dr. Adler given us something of the clinical history and laboratory results of the case. Igersheim has also reported a case in which the patient had syphilis.

Dr. Luther C. Peter said that it is difficult to determine from the fields alone whether this disease is primarily a disease of the choroid or retina. It is quite evident, however, that the retinal nerve fibers are caught up and evidently distributed by the lesion in this interesting type of chorioretinal disturbance. The lesion in the right eye, in the case which Dr. Adler presented, is up and to the temporal side, and, as one would expect, the field disturbance is down and to the nasal side, involving the fibers which are distributed to the superior temporal retina.

Jensen's disease undoubtedly differs from ordinary types of choroiditis in that, as Dr. Adler has pointed out, the sector from the disks to the periphery is involved, whereas in patches of choroiditis, field pathology usually

is limited to the area of choroidal involvement. A patient under his care, suffering from a patch of chorioretinal disturbance down and in from the disk, but not immediately adjoining the disk, showed the same field, disturbance extending from the disk to the periphery.

A Simplified Conception of Visual Field Changes in Chronic Glaucoma

Dr. Luther C. Peter called attention to the fact that, in accounting for the various field changes found in chronic glaucoma, three phases have not been analyzed to the same extent as those which are known under special names, such as Seidel's sign, Bjerrum scotoma, Roenne's step and Elliot's sign. These special signs can be regarded as a Bjerrum scotoma or one of its modifications.

The behavior of color response is peculiar to glaucoma; color suffering *pari passu* will form in the areas supplied by definite nerve bundles which are undergoing atrophy. Failure of green sensitivity is the earliest evidence of a beginning Bjerrum scotoma.

Moderate peripheral contraction is due to a stretching process of the long peripheral nerve fibers which, according to Fuchs, occupy the central area of the nerve head. In the peripheral contraction disturbance of the choroidal nutrition probably contributes little, if any, permanent defect. Disturbed retinal nutrition plays a larger rôle than that of choroidal disturbances, but the chief factor is the stretching process.

The occasional loss of a temporal quadrant or sector, as the first evidence of field cutting, is likewise due to the same stretching process.

An analysis of all these changes reveals the same pathologic process at work, namely, a stretching or compression of nerve fiber bundles. The area in the field in which the defect first appears is determined by the anatomic relation of the nerve head to the scleral ring, the area in the cribriform membrane, or supporting structure of the nerve fibers, which first yields to increased intraocular tension.

The field defect which is now regarded as characteristic of glaucoma is the Bjerrum scotoma. Other signs added from time to time are but modifications of this sign. All defects thus far observed in chronic glaucoma can be accounted for by the same process. As Elliot states: "The great underlying factor in chronic glaucoma is an interference with the nerve bundles, either on the optic disk or

as they emerge from it. Other factors are only contributory." It will reduce our conception of field pathology, therefore, to greater simplicity if the Bjerrum scotoma alone, in one of its various forms, be regarded as the characteristic defect. All forms of field pathology observed in glaucoma can be traced in their development to the sector type of atrophy, which is expressed in a Bjerrum scotoma.

Notes on Subconjunctival Cataract Extraction

Dr. Frederick Krauss described an operation for subconjunctival extraction of senile cataract.

The incision with the von Graefe knife, after the sclerocorneal section, is extended under the conjunctiva to near the fornix. The conjunctival flap is made loose, so that the lens may escape under it when the lens is removed either by expression or by the hook. The advantages are greater safety, simplicity, quick healing, freedom from the necessity of absolute rest on the back, no need of bandaging both eyes and a general increase in comfort on the part of both operator and patient.

Discussion. Dr. William Zentmayer said that he supposed that where this technic was employed, if one desired to do a preliminary iridectomy, the incision had better be made in the cornea, as we are all familiar with the difficulty of getting an ordinary conjunctival flap where a preliminary iridectomy has been made, because of the cicatrization at the seat of the scar.

Exhibition of a Portable Illuminated Test-card

Dr. J. Milton Griscom exhibited a portable test-card outfit which was designed for use in bedside or home refraction. The apparatus consisted of a stand with folding supports and a jointed upright, a cross-bar below to which a shaded electric light could be attached, and a second bar above to which a folding black screen was fastened and in front of which a folding test-card was hung. A perforated metal cap was provided to place over the light used for illumination, which converted it into a spot-light for muscle testing. The entire outfit could be detached, folded up and carried in a small bag.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

JANUARY 20

A Unique Complication of Tonsillectomy

BY DR. HORACE J. WILLIAMS

Dr. Williams reported the breaking of a tonsil-snare wire during tonsillectomy on a child, with escape of the smaller wire fragment. This fragment lodged in the right lower-lobe bronchus. The operation had been performed by a hospital interne. After localization by *x*-ray the wire was removed by Dr. Chevalier Jackson on the day after operation. A plea was made for careful inspection of all instruments, teeth, etc., before performing tonsil operations.

Acute Laryngeal Edema

BY DR. DAVID HUSIK

Dr. Husik classified laryngeal edemas as follows: (1) Acute inflammatory infectious type secondary to localized adjacent inflammation, or occurring during some infectious disease, such as scarlet fever, influenza or typhoid fever. (2) Non-inflammatory, non-infectious type caused by foreign bodies, burns, passive congestion, potassium iodide and angioneurotic conditions.

Two cases requiring tracheotomy were reported; one followed tonsillectomy, the other a peritonsillar abscess.

Discussion. Dr. Gabriel Tucker emphasized the necessity of routine laryngeal examinations. Where, however, marked inspiratory retraction of the suprasternal notch is present, low tracheotomy should be performed immediately, postponing more thorough examination until the patient has been freed from the dangers of asphyxia. Attention was called to

the subglottic form of laryngeal edema. Here the laryngeal and tracheal mucosa are involved. This type is liable to follow foreign-body aspiration, especially if that foreign body is a vegetable substance. In vegetable substance some especially irritative principle is apparently present. After bronchoscopic removal the edema frequently increases and a tracheotomy must be performed. The importance of careful postoperative care in tracheotomy was emphasized. Attention was called to the danger of giving even small doses of potassium iodide in laryngeal conditions. Such therapy often precipitates a dangerous laryngeal edema. In 33 per cent of the deaths following angioneurotic laryngeal edema, death was due to want of tracheotomy.

Dr. E. B. Gleason spoke on the occurrence of laryngeal edema in urticaria. Dehydration by croton oil is often of value in these cases.

Dr James A. Babbitt referred to a case of laryngeal edema that followed catheterization of the Eustachian tube. This case did not require tracheotomy.

Dr. Fielding O. Lewis cited 2 cases of acute edema of the larynx following vaccine administration in hayfever patients. Operation was not required. It was his feeling that the case reported by Dr. Williams was a timely warning to those doing throat operations to thoroughly inspect instruments before performing operations. A snare so devised that long wires are attached to the handle of the instrument would overcome the danger of snare-wire inhalation.

Dr. George B. Wood suggested the twisting of the free ends of the snare-wire.

Dr. George M. Coates endorsed this method. It can be used only with the softer varieties of snare-wire.

Dr. George Fetterolf spoke of the fragmentation of the cutting blade of a LaForce adenotome during operation. The fragments were all recovered.

Nonendoscopic Cases of Foreign Bodies in the Tonsil

BY DR. LOUIS H. CLERF

Attention was called to the frequency of the lodgment of fish bones, tooth-brush bristles, toothpick fragments, bony spicules, plant-stems, etc., in the tonsil crypts or substance. Those who wear dentures are more liable to this condition, as the sensitive oral mucosa is the natural protective mechanism in the detection of foreign bodies in the material taken into the mouth. When a foreign body has lodged in the tonsil the pain or discomfort is usually localized to that side, but may not refer directly to the tonsil. The diagnosis is made by inspection and removal accomplished without endoscopy. Sometimes a tonsillectomy is required when the foreign body has become embedded.

Our Present Knowledge of Procain, with a Brief Report of Experimental Carotid Injection of the Drug

BY DR. KARL MUSSER HOUSER

(By Invitation)

Dr. Houser's paper thoroughly reviewed the available literature on procain. Particular emphasis was placed upon the physiologic and pharmacologic properties of the drug. Attention was drawn to the work of certain investigators who had been able to save the lives of experimental animals that had been given lethal doses of procain, by artificial respiration, cardiac massage and adrenalin chloride intravenously. Artificial respiration and cardiac massage alone were unable to produce these results. In his own experiments he injected the internal carotid artery of dogs with double the procain dosage usually used in performing local tonsillectomy, in the attempt to learn whether the immediate distribution of fairly large doses of procain to the higher centers would cause death. None of the dogs so treated died.

Discussion. Dr. E. L. Eliason (by invitation) spoke of the widespread use of procain in general surgery. He recommended the use of a 0.5 per cent solution. This gives adequate anesthesia if the operator is willing to give it a little time. In the use of local anesthesia the phlegmatic individual is by far the best patient. Local anesthesia by infiltration should not be used in the neighborhood of infection. Abdominal operative patients have a less stormy convalescence when local anesthesia is used. Great care should be exercised in the preparation of the solution injected.

Dr. James A. Babbitt asked the influence of adrenalin on the toxicity of procain.

Dr. Houser stated that Easman, Erdman and Bonn had found that adrenalin chloride added to procain solutions increased the toxicity two and three-fifths times when tested intravenously on rabbits. This disadvantage, however, was offset, especially in tonsil work, by the delayed absorption and lessening of hemorrhage.

Atrophic Rhinitis—A Clinical Study: Preliminary Report

BY DR. A. J. WAGERS

(By Invitation)

Dr. Wagers reported upon 22 cases of atrophic rhinitis which had been systematically studied and treated. The average age

of these cases was twenty-five years. Headache was present in some degree in 11 cases. The sense of smell was lost in 6 and impaired in 10 others. Hearing was impaired in 6. In 11 cases there was evidence of sinus disease by transillumination. Sinus roentgenograms of 11 cases were taken; 9 of these showed sinus pathology. The blood usually showed a slight secondary anemia. The treatment given was two-quart irrigations with physiologic sodium chloride solutions three times daily. By this method symptomatic cures occurred in all cases; objective improvement of the atrophy was questionable.

Discussion. Dr. George M. Coates mentioned the paper of Kauffman, who maintained that in this condition maxillary sinusitis was always present. Also the idea of certain British observers, who considered acid-fast bacilli to give rise to the ailment. The Perez bacillus and treatment by vaccines made from this bacillus were discussed. Dr. Coates in his own investigations was unable to isolate this bacillus. In the cases investigated by him a Friedländer bacillus was found. Vaccine therapy with this bacillus gave temporary symptomatic relief.

FEBRUARY 17

Aural Investigation at Johns Hopkins Hospital

BY DR. S. J. CROWE

(By Invitation)

Dr. Crowe spoke of the work concerning aural investigation which is being planned and carried out at the Johns Hopkins Hospital. With the idea of correlating, on a large scale, ear function with microscopic findings, the following plan is being followed: All patients admitted to the medical wards are subjected to complete otolaryngologic examination. This includes objective findings of ear, nose and throat, and complete functional tests, including audiograms. The results of these examinations are carefully recorded, cross-indexed and filed.

Upon the death of a patient, if autopsy permission is available, the temporal bones are removed by an electric saw and prepared for the cutting of serial sections. The time between death and autopsy is carefully noted. In a special laboratory, serial sections are prepared, cross-indexed and filed. In time there will be available

a huge mass of postmortem material that can be readily correlated with the antemortem findings. The difficulties encountered in this work are many. It is often impossible to have the autopsy performed sufficiently soon after death to avoid postmortem changes. The technic of decalcifying the specimens and cutting of sections is not easy.

Another plan which is being followed has much to recommend it. Each year some investigator who has done outstanding work on the subject of the ear is invited to come to the University and to remain there for a year engaged in investigative work relative to some phase of the ear. A special fund makes this possible. Dr. Stacy Gaul, associate professor of anatomy of the University of Michigan, is at present filling this position.

Dr. Crowe's talk was illustrated with numerous audiograms and microscopic sections found in various ear conditions.

Discussion. Dr. B. Alexander Randall highly praised the program which Dr. Crowe outlined. In the opinion of Dr. Randall the value of the audiometer is questionable, because he feels that in transmission through the head phone some distortion of sound probably occurs.

Dr. Ralph Butler stated that in the treatment of ear conditions accurate knowledge of the pathology was so important that such studies as those undertaken by Dr. Crowe and his staff are of the utmost value. It was his wish that Dr. Crowe would return in several years and give in detail the results of this most vast work.

Dr. Crowe stated that all audiometer examinations were controlled by tuning-fork tests. There is much to be gained and learned from bone-conduction tests made with the audiometer.

MARCH 26

Some Phases of Streptococcic Infection in Acute Mastoiditis

BY DR. RICHMOND MC KINNEY

(By Invitation)

Dr. McKinney, on account of recent experiences with mastoid cases, has come to believe that the type of infection plays a much greater rôle in the prognosis and clinical symptoms than he formerly

thought. Three cases upon which he recently has operated were cited. All were children with symptoms of acute mastoiditis demanding operation. Each case showed a hemolytic streptococcus in pure culture. One showed positive clinical evidence of partial sigmoid sinus thrombois, with a blood culture positive for the same organism. Operation was postponed on account of the opinion of the bacteriologist, Mr. L. O. Dutton. Mr. Dutton claims to be able to foretell the results of streptococcic blood-stream infection with 100 per cent accuracy. He recognizes several types of streptococci existing as mixed strains, each of which is combined with a bacteriophage. When grown on artificial media this balance is disturbed and one of the other predominates. Conclusions are drawn from the manner of colony growth and the morphologic characteristics of the organism. He has observed cases where the bacteriophage predominated to clear up from a blood-stream infection in seventy-two hours. Where the organism predominated, the blood culture remained positive until a fatal termination.

Discussion. Dr. George M. Coates said that to him the prognosis value of the manner of growth of cultures obtained from mastoid wounds and the blood stream was a new idea. If this factor is constant in a large group of cases it will be of undoubted value in the estimation of the proper time for surgical interference. The organisms of epidemics have, in his experience, shown a tendency to be more virulent than the sporadic cases. In a series of hospital army cases Dr. Coates found that pneumococcic mastoiditis was much more difficult to control than the streptococcic variety. In civil practice many streptococcic cases are fulminant, but show a tendency toward self-limitation.

Dr. George B. Wood considered epidemic ear complications to be less severe than the sporadic cases.

Dr. James A. Babbitt stated that his tendency is to show more conservatism toward ligation of the jugular vein and interference with the sinus in thrombosis cases. He considered it most important that it be definitely established when to, and when not to, operate in these cases. The question of the value of intravenous dye-therapy was raised.

Dr. Fielding O. Lewis asked how many cases had been observed in the manner described.

Dr. McKinney stated that Mr. Dutton has reported a series of 10 cases of sepsis in which the prognosis was accurately made by the nature of organism growth. He, too, has a greater tendency toward conservatism when bloodstream infection exists. In some cases he has used intravenous dye-therapy with questionable results.

Aneurysm of the Aorta

BY DR. FIELDING O. LEWIS AND DR. C. J. BUCHER
(By Invitation)

Dr. Lewis was called to do a tracheotomy upon an adult male on account of dyspnea. Upon examination the classical signs of laryngeal obstruction were absent and the patient was removed to the hospital for study. Soon after his arrival the dyspnea became so marked that it was necessary to give relief if possible. Previous blood studies reported a slightly positive Wassermann reaction and *x*-ray examination showed no aneurysm. Dr. Louis Clerf was called in to introduce a bronchoscope. He was unable to enter the left bronchus and the right with difficulty. When this was done the patient breathed more easily. A Jackson cane-shaped cannula was then inserted, giving the patient some relief from dyspnea. The appearance was that of mediastinal neoplasm or new-growth in the lung. A later Wassermann reaction was strongly positive and roentgen-ray studies by Dr. Manges showed a definite aortic aneurysm. The patient died in some days from asyphyxia. Dr. Bucher demonstrated the specimen.

Serial Frozen Sections of the Thorax in Sternum-eroding Aneurysm of the Arch of the Aorta

BY DR. GEORGE W. NORRIS AND DR. GEORGE FETTEROLF

Apropos of the case reported by Dr. Lewis, Dr. Fetterolf demonstrated slides of 2 cases previously observed and reported by Dr. Norris and himself. Soon after death the bodies were formalized, frozen and massive sections made through the thorax. These sections definitely show the possibilities of pressure that may be exerted by aortic aneurysms. Full accounts of these cases may be found in the *Annals of Otology, Rhinology and Laryngology*, September, 1917, and *Archives of Internal Medicine*, July, 1920.

Discussion. Dr. Louis Clerf stated that it is unwise to do endoscopic procedures in cases of aortic aneurysm. Here the emergency demanded action, and previous observers had ruled out aneurysm.

Closure of Retroauricular Fistula

DR. JAMES A. BABBITT

Dr. Babbitt presented a fourteen-year-old girl upon whom he had performed a plastic procedure for the closure of a retroauricular fistula six years in duration. The opening was $\frac{1}{3}$ inch in diameter and had persisted after mastoid operation. He used a modified Troutman procedure with excellent cosmetic results.

APRIL 21

Aphonia: Report of Three Cases

BY DR. WILLIAM R. WATSON

Dr. Watson called attention to the relative infrequency of attributing aphonia to physical causes and the prevalence of the diagnosis of functional aphonia. Acute laryngitis, tuberculosis, syphilis, benign or malignant growths, paralysis of the intrinsic laryngeal nerves following neuritis or pressure on the laryngeal nerves from any cause, hysteria, cerebral lesions affecting the cortical centers governing laryngeal enervation and cerebral concussion were given as the most common causes of this condition. In addition, a case of carcinoma of the right tonsillar region, in a woman twenty-five years of age, was given. Surgical removal was accomplished with no evidence of recurrence to date.

Three illustrative cases of laryngitis were cited. In two tuberculosis was the cause; in the third, lues. The first case was of especial interest, for although the laryngeal lesion was definitely shown to be tuberculous by microscopic examination, no evidence of tuberculosis elsewhere could be found.

Reference was made to Mr. Herbert Tilley's report, in 1918, of 23 cases of unilateral laryngeal paralysis. A follow-up of these cases showed that 7 had completely regained normal function, 10 were not heard from, 3 were unchanged and 3 had died. It should be remembered that unilateral functional vocal-cord paralysis can occur.

Discussion. Dr. George B. Wood stated that he had seen 2 cases of unilateral cord-paralysis with complete recovery.

Dr. N. P. Stauffer spoke of the prevalence of aphonia in the recent epidemic of acute colds. At the present time he has an adult female under observation, suffering from aphonia, whose larynx is normal in appearance.

Dr. James A. Babbitt cited 4 cases of aphonia in his practice. In 1 the cause has not been determined. In another the condition followed traumatic cellulitis of the throat. A third was due to anteropoliomyelitis limited to the laryngeal enervation. The fourth followed thyroidectomy.

Dr. George Fetterolf agreed with Dr. Watson that primary tuberculosis of the laryngeal does not occur. It is comparatively easy for a small pulmonary lesion to escape detection even when our most advanced methods of examination are utilized. This lesion could caseate, break down, establish laryngeal disease, and later heal and so escape detection.

Sinus Disease in Children

BY DR. CHARLES R. HUGHES

(By Invitation)

The method of Dr. Hughes, in making a diagnosis of sinus disease in children, is to make an examination of the interior of the nose by reflected light and confirm or disprove the existence of ethmoiditis. The patient is then sent to the roentgenologist, whose findings are often of value. Sometimes this exposure cures a catarrhal ethmoiditis. It is of great importance to determine the presence of maxillary sinusitis. If antrum disease is present, the antrum is opened intranasally under gas. If this fails to accomplish results, a radical external operation is done.

Discussion. Dr. C. C. Eves stated that the importance of sinus disease in children is becoming more recognized in recent years. The removal of tonsils and adenoids does not always clear up this condition. Exposure to the roentgen ray often helps ethmoiditis. He sometimes gives as many as five roentgen-ray treatments.

Dr. N. P. Stauffer stated that he has seen several children die of nephritis probably caused by sinus infection.

Dr. Karl M. Houser (by invitation) spoke of the methods of diagnosis and treatment in sinus conditions of children used by Dr. Dean, of Iowa City.

Dr. Hughes was of the opinion that in acute suppurative sinusitis complicated by nephritis, wide drainage by surgical measures should be established.

Retropharyngeal Abscess Discharging through the Right Ear

BY DR. C. C. EVES

A TWENTY-MONTH-OLD child was admitted to the hospital suffering from extreme dyspnea and cyanosis. A short time later the child became comfortable and a profuse discharge was noted coming from the right ear. This was later proven to be a retropharyngeal abscess that had evacuated itself through the right external auditory canal at the junction of the cartilaginous and osseous portions. The middle-ear was not affected.

There are three routes this pus could have followed to reach the auditory canal: (1) Through the fissures of Santorini; (2) through the fibrous tissue between the junction of cartilage and bone, which is at times present, especially in children; (3) through a dehiscence of the bony canal, which is present in a small percentage of cases.

Dr. Eves cited several other cases of retropharyngeal abscess that presented grave symptoms. In one hemorrhage was sufficiently severe to require carotid ligation. In another severe meningeal irritation was present. All cases cited recovered.

DISCUSSION. Dr. George B. Wood cited a case of quinsy which evacuated through the external auditory canal.

Another case of retropharyngeal abscess discharging through the ear was mentioned by Dr. N. P. Stauffer.

Dr. George B. Wood mentioned a case of quinsy that ceased to breathe following the evacuation of a peritonsillar abscess under ether and was resuscitated with difficulty. No pus had entered the trachea. He feels that the sudden release of pressure is in some ways responsible for the failure of respiration.

Dr. James A. Babbitt spoke of a peritonsillar abscess in a child where slight pressure on the abscess caused failure of respiration and death. No incision had been made.

Dr. George Fetterolf had a patient stop breathing during the evacuation of a peritonsillar abscess. In this case it was due to suffocation. By removing the pus from the trachea by gravity, breathing was restored.

Dr. Stauffer cited the case of an adult male who suffocated following the spontaneous evacuation of a large peritonsillar abscess.

Dr. C. C. Eves called attention to the occurrence of meningeal irritation in retropharyngeal abscess. He also stated that the literature contains reports of fifty or more cases of fatal hemorrhage following the evacuation of peritonsillar abscess.

MAY 19

Joint Meeting of the Section on Otology and Laryngology of the New York Academy of Medicine and the Section on Otology and Laryngology of the College of Physicians of Philadelphia

The Management of Acute Suppurative Otitis Media in the Home

BY DR. FRANCIS R. PACKARD

In order to stimulate discussion and in the attempt to secure some unity of method in this condition, Dr. Packard presented the above subject. It was admitted by all that prompt and adequate incision of the tympanic membrane is advisable whenever there is distinct evidence of middle-ear abscess. The following home-treatment was outlined:

1. Early myringotomy.
2. Gentle instillation of cleansing solution and suction of ear secretion, using a glass pipette.
3. Drying of the ear following irrigation.
4. Most any sterile solution will do for cleansing, such as boric acid, normal saline, weak phenol, etc.
5. The use of heat for pain where the mastoid is not involved.
6. The use of cold where there is impending mastoiditis.
7. Secondary myringotomy if necessary.

Discussion. Dr. L. M. Hurd (New York) gives mimeographed instructions to his acute otitis cases. It is, he feels, important to apply heat and facilitate drainage by frequent cleansing. He accomplishes this by means of constant thermolite application, applied through a hole in a piece of cardboard so arranged that the face is protected from the light rays. The secretions are wiped away as they appear.

Dr. J. J. King complimented Dr. Packard on his presentation of this subject. It is his practice to irrigate with boric acid solution by means of a rubber bulb.

Dr. A. P. Voislawsky (New York) irrigates only when drainage is profuse.

Dr. George M. Coates condemned the common practice of cotton drainage wicks in the canal. In his opinion such practices tend to hinder free drainage. He uses no cotton. If it is used, it is to be changed every half hour. Attention to the nose and throat is most important. For this purpose argyrol, silvol, neosilvol or camphor-menthol solutions can be used.

Dr. E. V. Hubbard (New York) asked if any members had any experience with the Alpine lamp as a therapeutic measure.

Dr. McPherson (New York) favors the use of some hydrogen peroxide to prevent sealing of the myringotomy opening.

Dr. B. A. Randall stated that leeching has been abandoned on account of the large opening now made. If properly and early performed, leeching can be of great value. Atropine has considerable local analgesic value ($\frac{1}{20}$ grains against the tympanum). Aminol is very valuable in controlling pain. Phenol-glycerin solutions are beneficial on account of the analgesic and hydroscopic action. In his experience, heat has uniformly given better results than cold. Bandaging of the ears is important.

Dr. N. P. Stauffer was of the opinion that cold applications tended to limit the bacterial growth and in this manner exert a more beneficial action than heat.

Dr. Packard, too, felt that where mastoiditis threatens cold gives better results than heat.

A Contribution to Our Knowledge of the Sphenopalatine Ganglion

BY DR. GEORGE FETTEROLF,

DR. E. H. CAMPBELL AND DR. K. M. HOUSER (By Invitation)

Dr. Campbell first covered the bibliographic history of the sphenopalatine ganglion syndrome and its treatment. Eight cases that had received alcoholic injections of the ganglion *via* the posterior palatine canal were then reported. These cases have been carefully selected and the diagnoses established by means of a preliminary procain injection. All were given total or partial relief. The use of ganglion injections for local surgical anesthesia for intranasal work was reported favorably and a number of cases presented.

Dr. Campbell had dissected the ganglion and canal in 15 specimens. Each of these specimens had been injected with 1 cc. of 70 per cent alcohol stained with methylene blue. In his dissections he found that:

1. The thickness of the palatal tissue over the posterior palatine canal varied from 0.5 to 1.1 cm.

2. In some specimens the canal was large enough to permit lateral motion of the needle; in others the needle fitted snugly. In the former the ganglion was not equally stained, sometimes the outer and sometimes the inner being more deeply stained, depending

whether the needle had passed along the outer or inner wall of the canal.

3. In 9 of the 15 specimens the needle struck the bone 3.7 to 4.9 cm. from the palatal surface. This bone was always the antero-lateral part of the under surface of the sphenoid sinus. In 2 cases the needle point would slip off of this surface and penetrate the sphenomaxillary fissure.

4. The sphenopalatine foramen was found usually just above the posterior end of the middle turbinate. In some cases the turbinate extended beyond the ganglion, in others the ganglion was placed superior to the middle turbinate.

5. In 9 cases the needle could be passed beyond the sphenoid bone. In 8 of these cases the needle was found just internal to the maxillary nerve as it emerged from the foramen rotundum. In the other it was 0.2 cm. external to this foramen.

6. If the needle was inserted an average distance of 3.96 cm., it was found to reach the level of the Vidian nerve. Injection at this level always injected the Vidian nerve.

7. It 2 cases defects of the bony wall of the posterior palatine canal made it possible for the needle to enter the inferior nasal meatus.

8. In all cases but one the stain was found to have penetrated the ganglion. Where injection was made at 3.5 cm. the maxillary nerve was not stained. At 4 cm. the stain was found on the under surface of the orbital tissues. It came no nearer than 0.75 cm. to the optic or opthalmic nerves. At 4.5 cm. the maxillary nerve was deeply stained and just touched the external portion of the ganglion. All specimens showed some stain in the zygomatic fossa, it having gained this region through the pterygomaxillary fissure.

Discussion. Dr. S. Ruskin (New York) was delighted to find that his work had been checked in Philadelphia. He originally got the idea of injecting the ganglion from Silberman, a dental surgeon, who used this approach to the maxillary nerve. One should be impressed with the dangers of this injection. Not more than 10 to 12 drops of alcohol should be used. Phenol combined with alcohol is to be preferred. The patient should be told that the face may be numb. He has had one case of trophic ulceration and one of temporary abducens paralysis. The optimum distance for injection is 3.5 cm. He has relieved about forty different complaints by this injection. As yet we know comparatively little of the wide nerve ramifications and connections of Meckel's ganglion. It is considered quite likely that constant irritation of one of the ganglia of the

vegetative nervous system may give rise to vagatonia. This ganglion is the one most often exposed to unnatural conditions.

Dr. Jones (New York) spoke on the negative side of ganglion injection. One asthmatic was made worse by the injection. He cautioned against undue enthusiasm. When used for nasal operations the bleeding is more severe than when cocain is used. Local applications, in his opinion, are preferable.

Foreign Body Accidents during Tonsillectomy

BY DR. GABRIEL TUCKER

Dr. Tucker presented a series of 11 cases of aspiration of foreign bodies during tonsillectomy. The objects were mouth-gag lamp, piece of tonsil-grasping forcep, wire fragment from tonsil snare, tooth crown, deciduous teeth, portion of nasal rasp, gauze sponge and a cotton swab-stick. The diagnosis, symptomatology and prophylaxis of these conditions were considered.

Discussion. Dr. H. H. Forbes (New York) stated that we are fortunate in having a school of bronchoscopists who can handle these cases when they arise. He, too, feels with Dr. Tucker that blood-stream infection probably explains more lung abscesses than direct aspiration.

Dr. Fowler (New York) considers Dr. Tucker's presentation most admirable. In the last five years, in a clinic doing about 10 cases daily, they have had but 1 inhalation case. In this case a piece of tonsil lodged in the trachea. It was removed by suction.

Dr. E. V. Hubbard (New York) once had a tonsil-knife break during operation. He was able to remove the fragment before it was aspirated.

OCTOBER 20

Typhoid Ulceration of the Larynx

BY DR. JOHN R. DAVIES, JR.

In 1860, Barthey first called attention to the occurrence of laryngeal conditions in typhoid fever. Later, observations were made and recorded by many other writers. Most observers consider the condition rather infrequent. Fussell found that in 2000 cases,

in Munich, only 15 showed laryngeal involvment. Others reported similar infrequency. One notable exception was the series of cases reported by Jackson, who found laryngeal involvment in a much higher percentage of cases than any other observer.

The case was reported of a twenty-four-year-old colored man who developed a laryngeal lesion during the sixth week of typhoid fever. This subsided, but again became active in the ninth week, when tracheotomy was required. Dyspnea was not relieved and death followed in twenty-seven hours.

The autopsy revealed atelectasis of both lungs, due to blockage of the bronchi with mucus. Extreme swelling of the vocal cords entirely obliterating the glotic chink was found.

Discussion. Dr. J. A. Babbitt cited a case of laryngeal ulceration in which both arytenoid cartilages were coughed up. A fair speaking voice was retained.

Dr. George B. Wood spoke of a case of typhoid fever in a child, where a tracheotomy was necessary.

Dr. Louis Clerf referred to laryngeal stenosis in adults following typhoid laryngitis in childhood.

Dr. C. C. Eves recalled the case of an adult who, during typhoid fever, required tracheotomy. No typhoid bacilli were found in the laryngeal lesion.

Case Reports

BY DR. G. M. MARSHALL

Aural Exostoses. The case of a sixty-six-year-old adult who became deaf on account of closure of the canal by a bony growth. Recovery followed surgical removal.

The second case, an adult male, suffered from pain and deafness. An obstructive bony growth was found, which was removed. Pain was relieved but deafness persisted, as the patient had worked for years in a boiler factory.

Exposed Bone following Radical Mastoid Operation. A radical mastoid operation was performed two years ago upon an adult female. The interesting feature of this case was the bony condition of the anterior and posterior portions of the innermost extremity of the canal. The bone in this region was dark, had no epithelial

covering and had the feeling of necrotic bone. On account of the absence of symptoms no attempt at removal was advised.

Discussion. Dr. Robert J. Hunter stated that in about 2000 aural examinations in service men he had found a considerable number of exostoses. None demanded interference. Most of these are probably an unusually large tympanic spine at the notch of Rininus.

Dr. George Fetterolf referred to the frequency of exostoses of the aural canal in England. By some this is considered to be the result of cold bathing.

Dr. George M. Coates has seen many exostoses of varying size. So far, none have demanded surgical removal.

Dr. W. B. Davis referred to a case of long-standing suppurative otitis media in which Nature by bony necrosis had performed a complete mastoid operation.

Dr. G. M. Marshall, in closing, said that German observers found no exostoses of the aural canal in an examination of 1900 children. These exostoses seem to have a predilection for the outer portion of the bony canal and are probably the result of some irritative process. Examinations of the skulls of primitive races show that the condition was quite common. It is especially common in swimmers and divers.

Orbital Abscess Secondary to Ethmoidal Infection in Children: A Report of Two Cases

BY DR. WARREN B. DAVIS

The ophthalmic and rhinologic literature indicate that approximately 60 per cent of orbital abscesses are secondary to sinus infection, and the majority of these from the ethmoidal region, especially during childhood. Probably most of these abscesses are due to contiguity, but it is possible that in some cases phlebitis of ethmoidal veins or lymphatic infection may explain their occurrence. Two cases were reported.

The first, a twelve-year-old child, presented typical local and general symptoms of orbital abscess, with purulent discharge from the right middle meatus. Opening of the anterior ethmoid cells was attended by the escape of a large amount of pus. Complete recovery followed.

The second case was that of a five-year-old child who developed a left orbital abscess secondary to ethmoid infection. This, too, was completely relieved by operation on the ethmoid cells.

Discussion. Dr. George B. Wood cited 2 cases of orbital abscess secondary to sinus suppuration. One, following ethmoiditis, was relieved by operation. Another, in which the abscess followed perforation of the inferior orbital plate during empyema of antrum of Highmore.

Dr. Fielding O. Lewis referred to a case of abscess of the orbit in a child, secondary to ethmoiditis. This was entirely relieved by external operation.

Nasopharyngeal Cyst

BY DR. WILLIAM HEWSON

In removing the tonsils of a twenty-four-year-old female, under general anesthesia, a cyst about the size of an almond was found in the midline on the posterior wall of the nasopharynx. No adenoid tissue was present. On incision, thick pus escaped. The diagnosis of nasopharyngeal cyst, or cold abscess, was made. Thornwaldt's disease was considered, but the absence of pharyngeal tonsil made this diagnosis untenable.

Discussion. Dr. George Fetterolf considered that the condition could possibly be due to incomplete closure of the duct, which in embryonic life leads to the pituitary body.

Dr. W. B. Davis stated that dissections show that quite a few individuals retain some remnant of this duct.

Dr. J. Clarence Keeler removed a similar cyst five years ago. Recurrence followed removal.

NOVEMBER 17

Is Radium a Cure for Cancer of the Larynx? An Inquiry Based on a Study of the End-results

BY DR. THOMAS J. HARRIS

After a comprehensive review of the literature on the above subject, Dr. Harris reported a series of 60 cases of laryngeal neoplasm treated with radium; 30 of these cases were treated by Dr. Imperatori and 30 by Dr. Harris, at the New York Post-Graduate Hospital. In 2 cases cures were observed extending

nearly five years. They concluded from their work with radium that it is not a cure for cancer of the larynx. The use of emanation seeds is yet too new to properly estimate the results obtained by this method of treatment.

The danger of serious after-effects when radium is used in laryngeal neoplasm was discussed. Toxemia following extensive necrosis, stimulation of neoplastic cells and increase of dysfunction of the parts have all been observed following the use of radium in this condition.

Dr. Harris has seen not merely failures result, but suffering increased and exitus hastened. He, therefore, concludes that radium, to date, in the treatment of laryngeal cancer, is not only a disappointment but is also positively harmful. Never where there is choice should radium be substituted for surgery.

Discussion. Dr. H. H. Forbes stated that he has, in conjunction with Dr. Harris, used radium in laryngeal cases since 1908. In benign cases the results were good, but the results where malignancy was present were universally bad. Biopsy as a diagnostic measure was advocated.

Dr. Henry K. Pancost no longer uses radium in the treatment of laryngeal cancer except in special cases. Cancer of the larynx is unsuited to radium therapy, (1) because of the resistant nature of squamous-cell carcinoma, which is the type most often found; (2) because of the tendency of necrosis of the laryngeal cartilages after the intensive radiation necessary; (3) because of the frequent inaccessible position of the growth. Prior to 1924, Dr. Pancost had treated 30 cases with radium; only 1 of these was a proven case of squamous-cell carcinoma: 6 of these patients are now living. Surgery gives better results in laryngeal cancer than radium.

Dr. Fielding O. Lewis has found that the use of radium in laryngeal cancer makes subsequent surgery much more difficult. Since 1919 he has performed 85 total laryngectomies for both extrinsic and intrinsic growths. When the diagnosis is established and the growth can be completely removed surgery is the only necessary procedure. All type of laryngeal carcinoma except the intrinsic type involving the anterior two-thirds of the cord should be treated by total laryngectomy.

Dr. G. E. Pfahler stated that he had never seen a case stimulated and impaired after radiation; also that he was more enthusiastic than ever over the radium treatment of cancer. He cited a case considered surgically inoperable which recovered following radium treatment. Dr. Pfahler has not observed the toxemia following treatment referred to by Dr. Harris.

Dr. Harris, in closing, emphatically stated that he has seen laryngeal growths stimulated by radium application. The results obtained by Dr. Pfahler were decidedly different from their own in New York, and were possibly the result of better application technic. He has abandoned all radium therapy except the use of embedded platinum radium seeds, which

give promise but are too new to definitely estimate their value. On account of the unsuccessful results secured in New York in the treatment of laryngeal cancer by radium, there is a general sentiment in that city toward abandoning such therapy in this malady.

DECEMBER 15

Presentation of New Instruments

BY DR. GEORGE FETTEROLF

Dr. George Fetterolf demonstrated a new type of chisel for the removal of the bony ridge in submucous resection of the nasal septum.

Dr. George M. Coates showed an electric device for the application of vaporized iodine in the treatment of the Eustachian tube.

A new type of mastoid retractor, along with a case for the carrying of small knives, was demonstrated by Dr. Curtis C. Eves.

Unusual Types of Mastoiditis

BY DR. S. MAC CUEN SMITH

Except the occasional instance where mastoiditis develops secondary to trauma or metastatic infection, it is conceded that the disease is secondary to inflammation of the Eustachian tube and tympanic cavity. If the tube remains patulous and the membrana tensa only is involved, serious mastoiditis is unlikely. If the vault is invaded and communication with the lower portions of the tympanic cavity blocked by swelling, and if the process is a suppurative one, the mastoid is always involved. The development of surgical mastoiditis, especially when the vault is involved, depends on the absence of free drainage, the virulence of the infection, the resistance of the patient and the composition of the osseous structure.

When the mastoid cortex is sclerotic and thick, the danger of intracranial complication is greater than where it is possible for an external fistula to develop. Many cases do not present the classical text-book symptoms.

The continuation of fever and abundant discharge is a grave sign. The absence of fever does not, however, rule out surgical mastoid in the presence of otorrhea. Where symptoms of mastoiditis occur after the discharge has ceased, surgery is necessary. In some cases the tympanic membrane is little changed. In such cases the canal is usually swollen. Narrowing of the canal, when not due to furunculosis or otitis external diffusa, helps in the diagnosis of the surgical mastoid.

When a copious purulent discharge is present at the end of four or five weeks, even though other symptoms are absent, surgical mastoiditis is present. As a rule, when multiple myringotomy must be performed, surgery of the mastoid is necessary.

Four cases were reported.

The point of interest in the first case was that mastoid disease developed without a suppurative otitis media. Some redness of Sharpnell's membrane and of the long handle of the malleus was present, but no other indication of middle-ear disease. The few signs of mastoiditis over the mastoid process could be explained by the presence of furunculosis of the canal. The *x*-ray showed clouding of the mastoid, and operation revealed pus under pressure. The tube and middle ear were no doubt first involved, but had largely recovered.

A second case was operated on in which mastoid pus was found, with erosion of the sinus plate. This patient had his initial symptom—consisting of pain on the right side of the head of four days' duration—three months previous to surgical interference. This pain subsided, but returned in a month, with swelling posterior to the mastoid extending almost to the occiput. This condition again subsided, to recur much more pronounced two months later. In this case, too, mastoid infection occurred with little evidence of pathology of the tube or middle-ear.

A third case required operation of the mastoid where profuse discharge and pain failed to disappear after multiple myringotomy. No local evidence of mastoiditis was present. The cortex was found to be extremely thick and the dura exposed.

In a fourth case, otitis media and later surgical mastoiditis followed furunculosis externa without any tubal infection.

Discussion. Dr. George M. Coates stated that the mastoid case with the classical text-book symptoms is easy to diagnose. The difficult cases

are those where few signs and symptoms are present. Such atypical cases, without middle-ear involvement, were originally called primary mastoiditis. Dr. Coates believes that all these cases have had at one time middle-ear involvement, perhaps mild in character. Many cases have been seen by Dr. Coates that showed mastoid disease with no objective or subjective evidence of middle-ear disease. A careful history is of great value. Any acute respiratory infection is of significance. The tympanic membrane may show no signs. In the interpretation of roentgen-ray plates one should always bear in mind that previous middle-ear inflammation may have changed the picture. The presence of ear discharge is important. Those that discharge a long time, with fever, usually require surgery. The same is true where multiple myringotomy is required; sagging of the posterior canal wall or narrowing of the canal by inflammation is one of the most important signs. Sometimes the infection is confined to the anterior horn of the zygoma. Such cases may simulate mumps. Dr. Coates asked for information concerning the diagnosis of masked mastoiditis in infants showing gastrointestinal symptoms.

Dr. Mathew Ersner (by invitation) considers the red blood cell picture to be of some value in the diagnosis of surgical mastoiditis. Early in the disease typical obstructive deafness is present. Later the deafness is mixed. Loss of some of the high tones will occur. After-treatment by Politzerization is important.

Dr. Arthur Wagers (by invitation) spoke of a case of furunculosis that later developed otitis media. Myringotomy was necessary. Later surgical mastoiditis developed.

Dr. Karl M. Houser (by invitation) referred to the case of a baby, aged eight weeks, that had been cared for at the University Hospital. This child had a bilateral otitis media requiring myringotomy. The ears became dry and the child improved. Some weeks later, frequent vomiting and temperature elevation occurred, the ears were objectively negative and myringotomy produced no pus. So profound was the emaciation that in the absence of other findings it was considered justifiable to open the mastoid antra. In each a small amount of thick pus was found. Vomiting ceased immediately and there was temperature elevation for a long time following operation. The baby gained weight and eventually recovered.

Dr. Smith, in closing, stated that the unusual cases of mastoiditis frequently seem much less atypical and unusual when thoroughly digested. Reading of the roentgen-ray plates by the surgeon is helpful. If the posterior canal droops, operation is seldom avoided.

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

PAPERS READ DURING 1926

JANUARY 25

Dr. G. Jackson Chandlee (by invitation), "Patent Ductus Arteriosus."

Dr. John B. Deaver (by invitation), "Jaundice."

Dr. Thomas Fitz-Hugh, Jr. (by invitation), "Syphilis an Important Cause of Delayed Resolution in Acute Pneumonia."

MARCH 1

Dr. Simon S. Leopold, "The Treatment of Polycythemia with Phenylhydrazin."

Dr. Alvan LeRoy Barach, New York City (by invitation), "Oxygen Therapy in Pneumonia."

Dr. A. J. Cohen (by invitation), "Surgical Treatment of Pulmonary Tuberculosis."

MARCH 22

Dr. J. Cutler (by invitation), "The Graphic Presentation of the Blood Sedimentation Test."

Dr. Russell S. Boles (by invitation), "Extraordinary Dilatation of the Stomach Secondary to Malignant Obstruction of the Pylorus. Report of a Case."

Dr. H. Harris Pearlman (by invitation), "Ultraviolet Light as a Therapeutic Agent."

APRIL 26

Dr. Edward B. Krumbhaar and Dr. Joseph Sailer, "Prognosis in Partial Branch Bundle-block."

Dr. George W. Norris, "What is Accomplished by Stimulation?"

Dr. Charles C. Wolferth, "Observations on Pulsus Alternans."

Dr. James E. Talley, Dr. John Eiman and Dr. Joseph Sailer, "Coronary Thrombosis."

Dr. Thomas McMillan, "Electrocardiographic Changes Encountered in Acute Rheumatic Fever."

SEPTEMBER 27

Dr. Samuel Hadden (by invitation) and Dr. George Wilson, "The Use of Tryparsamide in the Treatment of General Paresis: A Report of a Series of 30 Cases on Treatment for Three Years."

Dr. Frederick C. Hill (by invitation), "Gastrocolic Fistula."

Dr. Foster Kennedy, New York City (by invitation), "Encephalitis."

OCTOBER 25

Dr. Edward Weiss, "Congenital Ventricular Septal Defect in a Man Aged Seventy-nine Years" (with Lantern Slides).

Dr. F. W. Saunderman (by invitation) and Dr. J. Harold Austin, "Changes in the Composition of Blood Serum in Lobar Pneumonia."

Dr. George I. Swetlow, New York City (by invitation), "Paravertebral Alcohol Block."

NOVEMBER 22

Dr. Jay F. Schamberg and Dr. Carroll S. Wright (by invitation), "Results in the Treatment of Lupus Erythematosus with Sodium and Gold Thiosulphate. Comments on Gold in Tuberculosis."

Dr. George Wilson, "Toxic Psychosis: An Analysis of 100 Cases."

Dr. William Herrick, New York City (by invitation), "Cardiac, Vascular and Renal Complications in Pregnancy: Their Relation to Some Problems in Medicine."

PROCEEDINGS

OF THE

SECTION ON PUBLIC HEALTH AND INDUSTRIAL MEDICINE

PAPERS READ DURING 1926

JANUARY 22

Dr. A. J. Lanza (by invitation), "Industrial Hygiene."

Dr. L. R. Thompson (by invitation), "Industrial Morbidity Statistics."

APRIL 19

Sir Arthur Newsholme (by invitation), "Some Remaining Problems in Maternity and Child Welfare."

MAY 24

Dr. Elton Mayo (by invitation), "Industrial Fatigue Studies."

Dr. W. J. McConnell (by invitation), "Health Service to the Smaller Industrial Plants."

Dr. Hubley R. Owen, "Medical Supervision of the Members of the Bureaus of Police and Fire."

DECEMBER 3

Dr. C. T. Graham Rogers (by invitation), "The Activities of the Bureau of Industrial Hygiene, New York State Department of Labor."

Dr. May R. Mayers (by invitation), "The Study of Industrial Poisonings."

Dr. S. J. Repplier and Dr. William G. Leaman (by invitation), "The Use of a Stock Vaccine in the Treatment of Colds."

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

Owing to the decision of the Council of the College only to publish the proceedings of the Section in abstract form, the programs of the various monthly meetings are appended below, with rather full abstracts of the Annual Students' Meeting only.

JANUARY 19

Dr. H. R. M. Landis, "Joseph Addison: Satire on Syphilis."

Dr. C. E. McClung, "Inheritance of Acquired Characters."

Dr. C. W. Burr, "Dr. S. L. Metcalf—A Forgotten Philosopher."

Dr. J. F. Schamberg, "Ravages of Smallpox among Royal Families of Europe." (Lantern.)

Election of Officers: Dr. David Riesman, Chairman; Dr. E. B. Krumbhaar, Clerk.

FEBRUARY 16

Dr. E. B. Krumbhaar, "A Series of Interesting Medals."

Lt.-Col. Fielding H. Garrison, U. S. Army, Surgeon-General's Office, Washington (by invitation), "Touch Pieces and Other Medals of Medical Interest from the Army Medical Museum." (By permission of the Curator.)

R. Tait McKenzie, (1) "Designing and Reproduction of Medals:" (2) Exhibition of medals from the collection of Dr. Henry Barton Jacobs, of Baltimore. Inspection of exhibits.

MARCH 29

Dr. J. H. Lloyd, "Dr. Bernard de Mandeville and his Fable of the Bees."

Dr. N. B. Gwyn, "The Masters of the Master—The Details of Inheritance and Influences as Seen in the Early Life of Sir William Osler."

Dr. T. Diller, "Some Pioneer Medical Men of Western Pennsylvania."

Dr. James Walsh, "Galen's Demonstration at Rome of the Ureterovesical Valve and that the Arteries Contain Blood Normally." (With lantern slides.)

APRIL 22

Dr. J. D. Aaronson (by invitation), "Comparative Pathology in the Talmud."

Dr. C. W. Burr, "Medical Words in Johnson's Dictionary."

Dr. V. W. Murray Wright (by invitation), "Surgical Experience and Observations in Russia and Turkey."

Dr. E. A. Schumann, "François Mauriceau and the Obstetrics of his Time." (Lantern slides.)

OCTOBER 28

Dr. E. B. Krumbhaar, "The Stigmata of St. Francis of Assisi."

Dr. C. F. Painter, of Boston (by invitation), "An Historical Study of the Development of Medical Schools."

Dr. F. Packard, "The Order of Hospitals of King Edward VI."

NOVEMBER 19

Prof. Geo. G. MacCurdy, Yale University (by invitation), "Most Recent Anthropological Discoveries." (Illustrated by means of lantern slides.)

COMMEMORATION OF THE CENTENARY OF THE DEATH OF LAENNEC

Dr. L. F. Flick, "The Life of R. Theophile H. Laennec."

Dr. H. B. Jacobs, Baltimore, Md. (by invitation), The various editions of Laennec's *Traité de l'Auscultation Mediate* were shown. Also an autograph consultation of Laennec's and an unique collection of Laenneciana. Items were from the College of Physicians' library and various private collections were also exhibited.

THE HISTORY OF SURGICAL ADVANCES IN THE PAST FIFTY YEARS*

By W. W. KEEN, M.D.

My first allusion, of course, is to Ambroise Paré, surgeon to five kings of France, whose skill saved his life in 1572 in the massacre of St. Bartholomew. He was the first to use the ligature for blood-vessels instead of the cautery or the boiling pitch to kill the poison in gunshot wounds. This he did because the supply of pitch gave out. He passed a sleepless night, because of his ligatures, but in the morning found the patients on whom he had used it in a far better condition than the others who had been "pitched" and cauterized.

My experience goes back to 1860, when I began my medical studies at the Jefferson.

We knew almost nothing of germs until 1876, when Lister came over to our International Congress of Medicine at the Centennial. I was convinced of the merit of his antiseptic surgery, and on October 1, 1876, as his first disciple in Philadelphia, I introduced the practice at St. Mary's Hospital and have never wavered in my allegiance.

Think of our poverty in resources and equipment! We did have ether (October 16, 1846) and chloroform (December, 1847), but we did not have any clinical thermometers until Sir Clifford Allbutt, of Cambridge, England, invented them in 1867, after the Civil War was over. We had used the ordinary thermometers for a few years, graduated with degrees and used in the axilla. We felt the patient's arm or neck, and, forgetting that our own sense of heat might not be normal, we said "no" or "slight," or "high fever." Think of a mother with several children having no thermometer to decide whether to rout you out of bed or not, and be grateful to Allbutt! Wunderlich published the first book on thermometry in 1868.

* Read December 17, 1926.

Our instrument cases were lined with fine, dust-gathering velvet, and we laid out our instruments on ordinary clean towels with only soap and water for them and for our hands.

Our ligatures were of undisinfected silk. One end was cut short and the other left long to be pulled on gently in four or five days to see if they had rotted loose. The larger vessels were indicated by a knot in the ligature, the largest by two knots. These were not pulled on for a week or so, and often, whenever no clot had been formed in the infected vessel, tearing them away was followed by a rush of blood. This had to be arrested sometimes by a ligature, a little above the divided end; sometimes by a new incision higher up.

Ten days after Gettysburg I was on duty as "Officer of the Day" for twenty-four hours, and during that night I had to arrest secondary hemorrhages five times. I have not seen as many cases of secondary hemorrhage from that date until now, due to Lister.

One of these cases I can never forget. Our hospital, with over 3000 beds, consisted of one-story wooden pavilions built *en echelon* with ventilators at the ridge pole. The bullet (a leaden, unjacketed, large, conical bullet) had entered just above the end of the left clavicle. It had not emerged. Its course—up, down, right or left, or directly backward—was unknown. The hemorrhage was copious, but not *very* copious. The only way to deal with it—for to enlarge the wound always increased the area of infection—was to try to pick up the end of the bleeding vessel by a tenaculum—a sharp hook—and tie it with undisinfected silk, and see if that stopped the bleeding. The only light I had was a square block of wood, with five augur holes bored partly through, and five candles in the holes. To enable me to see better, the holder of the light lowered it a bit, as the candles were rather long. Our general idea of any fluid, like water, was that it evaporated *upward*. The lamp-bearer forgot, or did not know, that ether vapor is heavier than air and falls *downward*. In an instant everything burst into flames; the etherizer flung the cone to his right and the ether bottle (we had no tin carriers in those days) to the left. Fortunately the bottle did not break. I instantly seized a towel and covered the patient's face, to prevent his inhaling the flames, and another to protect the wound. Meantime the hemorrhage continued. In a few minutes order was restored, and a third or fourth attempt with the tenaculum and a ligature controlled the hemorrhage. A number of us learned several good lessons that night.

The hypodermic needle was just beginning to be used. I doubt whether there were half a dozen of them, or half a dozen of thermometers, in the whole Army of the Potomac.

Once — but once only — I saw Gross try the sharpness of his knife by his finger, and, lifting his foot, he stropped it on his shoe-sole and stripped it clean (?) between his finger and thumb. The same revolving table was used for the cadaver at the lectures on anatomy and for the surgical operations.

We had a small so-called hospital, over a saloon next to the Jefferson, with ten or twelve beds in two rooms, one for men and one for women and children. As serious cases as removal of an upper jaw were sent home by carriage and the clinical assistants attended them there.

When Gross wanted to lecture on suppuration, I heard him regularly say to the orderly: "Hugh, tomorrow I'm to lecture on suppuration. Go to the hospital and get me half a tumblerful of pus," and the pus was always "on tap," even in the little hospital.

An amputation of the breast seldom was expected to heal under a month or more, for infection always was expected. Healing by "first intention" was bragged about for a month or more.

Erysipelas often set in and jeopardized the patient's life.

I often saw Gross and Levis "couch" for cataract, *i. e.*, inserting a needle and pushing the lens down, out of the line of vision, into the vitreous.

We never opened the head, the chest or the abdomen unless they had already been opened by accident.

"Hospital gangrene" — what irony in the adjective! spread from case to case in the Civil War, with a terrible toll of life. Amputation usually had a mortality of about 40 per cent.

The three days' fight at Gettysburg was seven times less dangerous than a compound fracture of the leg or a major amputation! So dangerous was a compound fracture of the leg that Syme, of Edinburgh, Lister's predecessor and also his father-in-law, advocated amputation above the knee as the less dangerous alternative.

I was the assistant to Dr. Washington L. Atlee, the best ovariotomist of his time, in the late sixties and early seventies. He never operated until compelled to do so by the size of the tumor. He saved only 1 out of 3 cases. Then, in 1876, came the glorious day of emancipation by the antiseptic method! Even that was decried as "only surgical cleanliness" by those who did not realize what

surgical cleanliness was. From then on, Lister's method was simplified. The spray was abandoned, for it constantly caused nephritis in the surgeon even more than in the patient, for he was inhaling the spray during several consecutive operations. Today the method is simplicity itself as compared with the cumbrous ritual of the seventies and eighties.

The rest is modern history. Even so late as 1889, however, when I was elected to the chair of surgery, there was not a single one-volume text-book based on bacteriology for me to recommend to my students as their *vade mecum*. So I said at once: "I will organize one." I selected twelve surgeons from the leading medical schools and I made the thirteenth –as if in bravado!—and in 1892 *The American Text-book of Surgery* was published and was a great success.

I congratulate you, my many friends, on your sumptuous heritage. Remember that you have entered into the heritage which the men of my generation had at first to grope for in the dim darkness, but ever with increasing light, developing into the splendid surgery of today.

See to it that, in your turn, you carry on the torch of learning to a better and brighter day than we have ever known!

THE HISTORY OF THE ADVANCES IN THE MEDICAL AND SURGICAL SPECIALTIES*

AN ABSTRACT

By GEORGE E. DE SCHWEINITZ, M.D.

THE necessity and value of proper specialization in medical and surgical work was first referred to. A brief description was then given of the "specialties" as they existed among Egyptian physicians, according to Herodotus; of the attention paid to ophthalmic work as recorded in ancient writings and books, for example, the Code of Hammurabi, the Ebers Papyrus, the volumes of Avicenna and of Aetius of Amida, and, in the Middle Ages, of the compilation of George Bartisch.

Taking ophthalmology as an example of an established "specialty" the following outstanding events in its evolution, were sketched:

1. The utilization of lenses (spectacles) as an aid to vision, with their history from the time of Roger Bacon's (1214-1294 A.D.) studies of convex lenses, to the discovery of astigmatism by Thomas Young (1801), and its first correction by cylindrical lenses by the Royal astronomer, Airy (1857), and soon after by Isaac Hays in Philadelphia.

2. The appearance of von Helmholtz's great work on *Physiologic Optics* (1856) and that of Donders on *The Refraction and Accommodation of the Eye* (1864), and his explanation that "asthenopia" or "eye-strain" was due to the muscular strain caused by hyperopia, and the recognition by Weir Mitchell, in association with William Thomson and Ezra Dyer (1872), of the enormous influence of refractive error and of "insufficiency" of the ocular muscles as a cause of headache, many nervous and mental phenomena, and functional disturbances—a discovery which deserves to rank with the best scientific announcements of the nineteenth century.

3. The invention by von Helmholtz of the ophthalmoscope (1851), whereby was established what amounted to a new science, namely, ophthalmoscopy, medical and neurological.

* Read December 17, 1926.

4. The development of ophthalmic surgery, prefaced by a brief history of the surgical procedures practised by ancient and medieval ophthalmologists, special attention being paid to the operation for the extraction of cataract, first performed by Jacques Daviel (1748 and 1752); the operations for acute glaucoma introduced by von Graefe in the form of iridectomy (1852), and the modern methods of establishing a permanent fistulous area; the great improvements in the technic of correcting various types of strabismus and the improved technic of blepharoplasty, etc.

5. The improvements in the instruments of precision and their use; modern ophthalmoscopy, quantitative perimetry, retinoscopy (shadow-test), and *biomicroscopy*, that is, thanks to Gullstrand's outstanding studies in optics, the development of slitlamp investigation, which well-nigh ranks in importance with ophthalmoscopic work.

6. The doctrine of focal infection in its relation to the cause and treatment of many ocular disorders, especially those of the uveal tract; protein therapy; and biochemistry in ophthalmology, which bids fair to achieve therapeutic results of the highest importance.

The intimate association of ophthalmology and neurology and neurologic surgery was also referred to, together with outstanding value of ophthalmic investigation in neurologic diagnosis and the advances in otology and laryngology and rhinology. The importance of coöperative work between the various specialties, as well as that which should exist between these specialties and general medicine and surgery was emphasized, and certain historical data which indicate that even Hippocrates was acquainted with the release of increased intracranial pressure by trephining as a means of restoring vision, were included.

THE HISTORY OF ADVANCES IN MEDICINE IN THE LAST FIFTY YEARS*

By O. H. PERRY PEPPER, M.D.

ΠΑΝΤΑ ΡΕΙ, all things flow, wrote Heraclitus, and H. G. Wells, in a recent book, applies this thought to the steady flow of human affairs and to the history of mankind. Change is an inherent and essential characteristic of existence, as true for the race and species as for the individual. We tend to be amazed at change, whereas it is an apparently unchanging state which should excite our interest as an unaging individual would. Our minds should be adapted to the concept of constant and progressive change in all living things. Change is ever-present, but the existence of change does not necessarily imply that the change is for the better. Change is not always progress.

Tonight we are attempting to judge the change in medical knowledge and science within the past fifty years; to estimate the direction of its flow, its rate, its results. It is far easier to judge such matters over longer periods and at a greater distance. We can today estimate the effects of Harvey's discovery; we can judge the ebb of knowledge during the dark ages. But the past five decades are too close, our judgment is warped and fallacious. Some items which we today consider very important will soon be forgotten; others which we scarcely notice will prove to be pregnant with unforeseen results.

In the early years of the period we are considering, there were still alive many great figures whose achievements were not accomplished within this span of fifty years. Stokes, Corrigan, Graves, Skoda, Rokitansky, Oliver Wendell Holmes, Semmelweiss, Broca, Darwin, Huxley, Helmholtz, Claude Bernard, Virchow, Long and Leidy. They died less than fifty years ago, and it was their influence which

* Read December 17, 1926.

brought medical knowledge to the level at which it was at the beginning of the period we are considering.

Let us turn back to the early days of this period and try to learn of the medical interests of the internist of that day and try to recapture a little of his point of view. We will at one moment be amazed at what seems to be our progress since that time; at another moment equally amazed at our failure to add to their clinical knowledge. Often we will find in their knowledge the germ of ideas we consider very new and revolutionary.

William Osler was in Philadelphia from 1884 to 1889 –about forty years ago. His interests of those days should help us visualize the times. Just forty years ago he read a paper to the Philadelphia County Medical Society "On the Use of Arsenic in Certain Forms of Anemia." This sounds familiar enough, and the next year followed articles on the treatment of pneumonia and the treatment of rheumatism. Osler was still far from the therapeutic nihilist he was later said to be. In 1888, came his famous lectures on the "Cerebral Palsies of Children;" articles on smallpox, duodenal ulcer, angioneurotic edema, and, not to be overlooked, a note on "An Accouchement in a Railway Closet." In 1887, Osler was very skeptical of the nature of the organisms described in 1881 by Laveran in malaria, but within the year he reported to the Pathological Society of Philadelphia his studies of these organisms in 52 cases of malaria. Two years later, only thirty-six years ago, he is still cautious in accepting the findings as "peculiar to and diagnostic of the presence of the malarial poison."

Cholera, yellow fever, malaria, typhoid and smallpox bulked large in the interests of the physician and pathologist in those days. This has changed, with no thanks to the antivaccinationists. Many new or apparently new interests occupy us today.

Let us open the first American *System of Medicine*, published forty-one years ago, in 1885, and seek for insight into the medicine of that period. Austin Flint writing on "Pulmonary Phthisis" states that Koch's recent discovery of the tubercle bacillus justifies the conclusion that phthisis is an infectious disease. Not a new theory by many centuries, for Plato maintained that phthisis was infectious, but proved only forty-four years ago.

In the section on "Valvular Heart Disease," the importance of rheumatic fever was stressed, but little mention was made of syphilis as a causative factor. Syphilis, indeed, was not included among

the causes of aortic insufficiency. An amusing argument is seriously presented: The frequency of heart disease among soldiers had apparently been used to support the relation of syphilis to heart disease, but the author points out that sailors are notoriously more subject to syphilis than soldiers and yet heart disease is rare among sailors. The correct explanation, he states, is to be found in the tight clothes of the soldiers and the loose clothes of the sailors.

Before we smile too condescendingly, let us remember that this was before the days of the Wassermann test or the discovery of the spirochete of syphilis. Also, let us keep in mind that Fournier and Erb did solve the relationship of syphilis to tabes without knowledge of the spirochete or complement-fixation.

No attempt at classification of the cardiac arrhythmias is given in this *System of Medicine*, but a pulse-tracing from a case of mitral stenosis is illustrated which to our eyes is clearly one of auricular fibrillation. No such concept was in their minds, the string galvanometer was needed to clarify that subject.

D. Hayes Agnew wrote the section on "Diseases of the Thyroid," which organ was supposed to regulate the supply of blood to the brain and also to supplement the work of the spleen. The thyroid gland was considered an appendage of the vascular system. One is amazed to find in Agnew's article no discussion of exophthalmic goiter or Graves' disease, but on more careful search a full description of this condition is discovered – not in the section on "Diseases of the Thyroid," but in that on "Neuroses of the Heart!" Again, our smile is premature, for there is a full discussion of the value of iodine in simple goiter, and it is suggested that it could be given in food, as, for example, baked in bread. Our present-day iodized salt is no great improvement or innovation. Surgery of the thyroid was in its infancy and only resorted to in direst need, as when suffocation threatened. Only seven years previously, Kocher had first excised a goiter.

It would be easy to multiply such examples of the great differences in medical knowledge or point of view today and fifty years ago. But we must beware lest an insidious sense of superiority takes possession of us.

Karl Sudhoff, the greatest medical historian of all times, describes in one of his inimitable sketches on medical history the various ways in which individuals react to the question, What is history of medi-

cine? He writes: "One man will shrug his shoulders and superciliously speak of an old curiosity shop; another will call it a catalogue of exploded theories; to another, who has had just a peep of it, it will offer a convenient opportunity for throwing out his chest and telling a gaping audience how wonderfully we moderns have advanced and what poor sticks our colleagues of yore have been, fellows without vision, without critical judgment, without method, so quite unlike modern science with all its profundity and inherent exactitude—in brief, to him the history of medicine will be a ready means for glorifying the present state of affairs and for belittling the men of the past 'as they well deserve.' And a third will use the history of medicine for launching a powerful speech with a few grains of knowledge and much phantasy, and if he wields a clever pen he can be as brilliant as he likes: hardly one in ten thousand can control the urge!"

Let us try not to fall into either of these faults and let us admit, once and for all, that the doctors of fifty years ago knew much and accomplished greatly with the wisdom that was theirs. Second, let us remember that change is not always progress, and that much of our vaunted knowledge of today may turn to ashes in our mouths within another fifty years. Third, let us realize that almost all our real progress has come from the application of some method previously unavailable or unutilized.

For example, bacteriology is wholly a product of a new method; the early years of our fifty-year period are dotted with the discoveries of important pathogenic organisms—the gonococcus by Neisser in 1879; the tubercle bacillus by Koch in 1882; meningococcus by Weichselbaum in 1887. These were triumphs for a new method—no wonder Pasteur enthused over the first culture plate exhibited.

In 1889, the proof of the relation of the pancreas to diabetes mellitus by von Mehring and Minkowski; the proof of the formation of sugar from protein in the diabetic organisms by Lusk eighteen years later; the importance of ketogenic-antiketogenic balance in the dietetics of diabetes by Schaeffer, and still more recently the discovery of insulin by Banting and his coworkers.

Ehrlich is quoted as saying that he never could have produced salvarsan if Schaudinn had not discovered the spirochete of syphilis four years before. Each step is made possible by a previous one and in turn makes another possible.

Let me now name one outstanding advancement of each of the fifty years of the period we are considering:

1876. Use of stomach tube—Ewald.
1877. Relation of liver to metabolism by Eck fistula—Eck.
1878. First excision of goiter—Kocher.
1879. Gonococcus discovered—Neisser.
1880. Parathyroid described—Sandström.
1881. Discovery of malarial parasite—Laveran.
1882. Discovery of the tubercle bacillus—Koch.
1883. Conception of body as machine—Voit.
1884. Theory of phagocytosis—Metchnikoff.
1885. Prevention of hydrophobia—Pasteur.
1886. Relation of pituitary to acromegaly—Pierre Marie.
1887. Meningococcus discovered—Weichselbaum.
1888. Studies on toxins—Roux and Yersin.
1889. Proof of relation of pancreas to diabetes—von Mehring and Minkowski.
1890. Diphtheria treated with antitoxin—Behring.
1891. Lumbar puncture introduced—Quincke.
1892. Discovery of Bacillus aërogenes capsulatus—Welch.
1893. Increased metabolism in exophthalmic goiter—Müller.
1894. Introduction of *x*-rays—Roentgen.
1895. Neurological studies of Charcot.
1896. Diagnosis of typhoid by bacterial agglutination—Widal.
1897. Researches on proteins, purins—Emil Fischer.
1898. Differentiation of human and bovine tuberculosis—Theobald Smith.
1899. Chemical activation of sea urchin egg—Loeb.
1900. Studies on yellow fever—Reed and Carroll.
1901. Parasite of sleeping sickness—Dutton and Ford.
1902. Isolation of adrenalin—Takamine.
1903. String galvanometer introduced—Einthoven.
1904. Respiration calorimeter introduced—Atwater.
1905. Discovery of Spirocheta pallida—Schaudinn.
1906. Discovery of Wassermann reaction—Wassermann.
1907. Sugar from protein in diabetes—Lusk.
1908. Introduction of duodenal tube and bucket—Einhorn.
1909. Discovery of salvarsan—Ehrlich.
1910. Use of digitalis in fibrillation—Mackenzie.
1911. Beri beri vitamine isolated—Funk.

1912. Calculation of blood reaction from CO_2—Henderson—Hasselbach—Lundsgaard.
1913. Test of immunity to diphtheria—Schick.
1914. Classification of arrhythmias—Wenckebach.
1915. Studies on spirochetosis icterohemorrhagica—Inada.
1916. Bronchoscopic advances—Chevalier Jackson.
1917. Introduction of the gas burette—Van Slyke.
1918. Introduction of quinidine—Frey.
1919. Advances in colloid chemistry—Loeb.
1920. CO_2 and intracellular reaction—Jacobs.
1921. Insulin—Banting and coworkers.
1922. Lipiodol—Sicard.
1923. Application of colloid chemistry to Mammalian blood—Van Slyke—Wu—McLean.
1924. Scarlatinal antitoxin—Dochez and the Dicks.
1925. Cholecystography—Graham.
1926. Parathyroid extract—Collip.

No two individuals would name the same list, for in many years there are several noteworthy contributions to decide between. This is merely my personal selection, and it will serve to remind us of the many diagnostic and therapeutic tools which we have at our disposal today and which the physicians of fifty years ago lacked. How much better should our diagnosis and treatment be than theirs was!

We are, as a whole, much the same in bodily and mental equipment as those of fifty years ago and those of fifty years to come. The level of medicine depends rather upon the methods available and our utilization of them. The statistical method of Louis, the auscultatory method of Laennec, are part of our equipment, just as are the newest immunological and chemical methods.

New methods are not always appreciated, for example, skin-testing for allergic sensitization was done in 1863, but only adopted after many years of neglect. A method applicable for medicine is often introduced in another field of scientific activity and must wait for some alert student of medicine to see its medical usefulness. Only one with a wide knowledge of medicine and with the proper training and background can apply a new method with the fullest success. If a new method be inaccurate or faulty the whole pack may go off on a false scent as, for example, when thirty years ago a poorly devised method for blood sugar led to the discovery of a

nonexistent substance named "jecorin," and the erroneous and very misleading conclusion that the sugar in the blood is not increased in diabetes mellitus. Only after advances in colloid chemistry had come was the error rectified.

Great advances have been made in medicine in the past fifty years, but just as great will be made in the next half century. In the making of these each of you will unconsciously play your part. Every day, by a process of trial and error, new methods, new applications, new theories are being tried out, accepted or rejected. If it is difficult to picture the flow of progress over fifty years, how much harder must it be to judge the progress from year to year? And yet, if we are to play our part in the advance, we must do this. Change will come year after year, and to keep up with the stream you must keep yourself keenly aware of the forward flow of medical science and art. If you do not, the backwaters, side eddies and reverse currents will lure you into some peaceful, perhaps, but stagnant calm.

To do our share we must keep out in the stream; we must advance with our chosen branch of knowledge. We must give honor to the achievements of the past fifty years, but we must pledge ourselves to even greater achievements in the fifty years to come.

THE HISTORY OF LABORATORY RESEARCH IN THE PAST FIFTY YEARS*

By JOHN A. KOLMER, M.D.

Fifty years ago Virchow was at the height of his remarkable career, had already finished his epoch-making work on embolism (1856), and his *Cellular-Pathologie*, published two years later, with its doctrine of "omnis cellula e cellula," placed descriptive pathology for the first time on an exact and firm scientific basis. One of his pupils, Julius Cohnheim, in 1867–1873, revolutionized the pathology of inflammation and suppuration by demonstrating the diapedesis of leukocytes in direct opposition to the teachings of Virchow, introduced the method of freezing fresh tissues for histological study and blazed the trail of experimental histology and pathology. Pathology, therefore, was on a firm basis prior to 1876 as the result of the labors of these two men; but the past fifty years have not been without additional notable discoveries in this field. At the beginning of this epoch Weigert, for example, had just finished his memorable work on the pathology of smallpox (1876), followed a few years later by that on nephritis (1879), the introduction of anilin dyes for the staining of bacteria (1875), studies in coagulation necrosis (1880) and the promulgation of his famous quantitative or overproduction "law" in 1882, showing that the amount of repair in an injured tissue is always in excess of what is needed and the basis of Ehrlich's side-chain theory of immunity. At about the same time Waldeyer was doing his work on the development of cancer (1872), Pepper (1875) had discovered the bone-marrow changes in pernicious anemia, and Welch (1877) was engaged in his pioneer work on acute edema of the lungs, embolism and thrombosis. It was the age of pathology, and thanks to the labors of these and other pioneers the subject held the place of first interest at the time and served to place medicine upon a firmer scientific basis. In the

* Read December 17, 1926.

early eighties, however, the theater of interest switched to bacteriology, public hygiene and immunology, but noteworthy contributions to pathology continued to be made by von Recklinghausen and Grawitz on tumors (1882-1884), Reginald Fitz on appendicitis (1886) and hemorrhagic pancreatitis with fat necrosis (1889), and by numerous others abroad and in the United States, among whom I need but mention such names as those of Barlow, Guitéras, Councilman, Flexner, Hektoen, Riesman, Stengel, Kauffman, Adami, Delafield, Prudden, Aschoff, Mallory and MacCallum.

But the past fifty years are particularly notable for epoch-making discoveries in bacteriology, immunology, public hygiene and allied subjects. Indeed it is almost impossible to properly estimate their influence upon medicine and surgery and the many and great blessings they have conferred upon humanity. It is true that some of the larger pathogenic fungi and parasites had been already discovered because falling into the range of vision of the microscopes then available; and for the same reason Casimir Davaine had already seen the huge anthrax bacillus in 1863. It is also true that Pasteur had already laid to final rest the arguments on spontaneous generation, established the foundations of his great work by discovering the nature of fermentation by bacteria in 1865 and its prevention by partial heat sterilization (since of world-wide renown in relation to the partial sterilization of milk under the name "Pasteurization"), followed by his discovery of the cause and prevention of silkworm disease (*pébrine*).

In 1874 he had received the now world-famous letter from the generous Lister, from which the following may be quoted: "Allow me to take this opportunity to tender you my most cordial thanks for having, by your brilliant researches, demonstrated to me the truth of the germ theory of putrefaction, and thus furnished me with the principle upon which alone the antiseptic system can be carried out. Should you at any time visit Edinburgh, it would, I believe, give you sincere gratification to see at our hospital how largely mankind is being benefited by your labors. I need hardly add that it would afford me the highest gratification to show you how greatly surgery is indebted to you."

But in 1877 his great discoveries in anthrax and chicken-cholera were made, followed in a few years by those in the etiology of malignant edema, boils and pneumonia, and when, in 1881, he discovered a practical method of vaccination against anthrax of

the lower animals, followed in 1885 by the successful immunization of little Joseph Meister against the dreadful rabies or hydrophobia, the whole foundation of modern active immunization was laid and the incalculable value of Jenner's basic discovery of preventive inoculation greatly strengthened and broadened.

In the meantime the star of the humble German country doctor Robert Koch was on the horizon through his work in anthrax in 1876, followed in 1877 by the description of new methods for studying and photographing bacteria, and in 1878 by his great paper on "Die Aetiologie der Wundinfektionskrankheiten;" but in 1881 he made the greatest single discovery in bacteriology up to that time, not in the nature of unraveling the etiology of another disease, but by the discovery of so humble a laboratory procedure as the gelatine-plate method for isolating bacteria, which up to that time had baffled even the great Pasteur, and which led the latter to embrace the modest Koch with the statement: "C'est un grand progrés." At the same time Koch pronounced his now famous four postulates, followed in 1882 by his discovery of the tubercle bacillus, in 1883 by the bacillus of cholera and of acute contagious conjunctivitis. In 1890 he discovered tuberculin and likewise made one of the few mistakes of his remarkable career when, under pressure from the German government, he prematurely announced it as a cure for tuberculosis, but the disappointment of which was partially neutralized by his discovery of its diagnostic value in this disease. In passing, mention may be made of his second mistake in announcing, in 1900, that the human and bovine types of tubercle bacilli—which in the meantime had been separated and studied by Theobald Smith in 1898, and the bovine organism having been for the first time actually isolated from the lymph glands of a child by Ravenel in the Pepper laboratories of the University of Pennsylvania—were not identical and that human beings could not be infected with the latter. In the meantime Hansen, in 1874, described the bacillus of leprosy, and Neisser, in 1879, the gonococcus, so that the foundations were laid for that part of our knowledge of bacteriology which every medical student can be relied upon to remember, namely, the postulates of Koch that the tubercle bacillus is acid-fast and that the gonococcus is Gram-negative.

With these great discoveries by Pasteur and Koch, a regular "debauch" of additional discoveries in bacteriology followed, and

especially in the era from 1880 to 1897, when most of the pathogenic organisms were isolated, and among which may be mentioned the typhoid bacillus by Eberth and the pneumococcus by Sternberg, in 1880; the bacilli of glanders and swine erysipelas by Loeffler, in 1882 to 1886; the diphtheria bacillus by Klebs and Loeffler, in 1883; the tetanus bacillus by Nicolaier, in 1884; the colon bacillus by Escherich, in 1886; the coccus of Malta fever by Bruce, and the meningococcus by Weichselbaum, in 1887; the bacillus of influenza by Pfeiffer, and the white staphylococcus of the skin by Welch, in 1892; the bacillus of bubonic plague by Kitasato and Yersin, in 1894; the dysentery bacillus by Shiga, in 1897; the whooping-cough bacillus by Bordet and Gengou, in 1906; the filterable viruses by Loeffler and Frosch in 1898, and, last, of the bacteriophage by Twort in 1915 and independently by d'Herelle at about the same time, ending with the discovery of the bacillus of tularemia by Francis a few years ago.

The past fifty years have been also rich in investigations in parasitology of no lesser importance. Ushered in by the discovery of the spirillum of relapsing fever by Obermeier, in 1873; of trypanosomes, by Lewis, in 1878; and of Filaria bancrofti, by Manson, in 1879, the following year saw Laveran's great discovery of the malarial parasite. During the next fifteen years several of the pathogenic trypanosomes were discovered by Evans, Bruce, Rouget, Elmassian, Dutton and Castellani; Leishmann and Donovan made their discoveries in kala-azar, Theobald Smith of piroplasmosis of cattle, Allen J. Smith and Charles W. Stiles of the American hookworm, which opened up such a wonderful opportunity for the Rockefeller Board to help countless thousands of the infested in southern countries and our southern states; the glory of this period being finally crowned by Schaudinn's discovery of the spirochete of syphilis, in 1905, followed in the same year by Castellani's of the spirochete of Frambesia tropica. Since then, numerous additional spirochetes have been discovered, especially of the leptospira of infectious jaundice, by Inado and Ido, in 1914, and of yellow fever, by Noguchi, in 1919, and methods for their artificial cultivation by Schereschewsky abroad and by Noguchi in America.

Needless to state, these discoveries in bacteriology and parasitology proved a wonderful stimulus to investigations in public hygiene and preventive medicine, many of them opened up avenues for rapid bacteriological diagnoses, disclosed the mechanism of the

transmission of the etiological agents and led to the evolvement of personal and public methods of prevention of infection. When Manson, in 1879, discovered *Filaria bancrofti* in mosquitoes, the way was opened for establishing the general law of metaxenia, concerned with parasites as intermediate hosts, which was followed in 1888 by the discovery, by Smith and Kilbourne, of the transmission of Texas cattle-fever in ticks; of malaria in mosquitoes by Ross, in 1889; of yellow fever in mosquitoes by Reed and his associates, in 1900; of African sleeping sickness in tse tse flies by Bruce, in 1894; of Rocky Mountain fever in ticks by Rickitts, in 1907; of Mexican typhus fever in the body louse by Rickitts and Wilder, in 1910, etc. Who can properly estimate the incalculable value of these discoveries to countless millions of human beings? Mysteries in the causation of dreadful diseases melted away and opportunities were created for such geniuses in public hygiene as Sternberg, Vaughan and Gorgas.

In the meantime great discoveries were being made in natural and acquired immunity and resistance to the infectious diseases. Ushered in by the observations of Panum, in 1874, and of Roser, in 1881, on the destruction of bacteria by body cells, great impetus was given to these subjects by the investigations of Metchnikoff in 1884, which led Lord Lister to state in 1896: "If ever there was a romantic chapter in pathology, it has surely been that of phagocytosis." Theories in immunity have come and gone but the work of this great man has remained, and while other have corrected some of his errors, yet the phenomenon of phagocytosis is more firmly established than ever as a mechanism of fundamental importance in immunity.

With the discovery of bacterial toxins by Brieger, in 1888, and of the antibacterial properties of the blood by Fodor, in 1886, and by Buchner, in 1890, the way was opened for starting an argument with Metchnikoff on the relative importance of the humors *versus* the body cells in immunity, and resulting in the discovery of diphtheria antitoxin by von Behring and of tetanus antitoxin by Kitasato in 1890, followed by that of the antivenens by Calmette, in 1894. It also induced the chemist Ehrlich to enter the lists, and resulted in the promulgation in 1897 of that bugbear of medical students, his side-chain theory of immunity, which, despite its obvious complexity and shortcomings, had the merit of stimulating a great deal of fruitful research in humoral immunity.

Indeed the decade from 1890 to 1900 may be properly designated as belonging to immunity, because of the numerous and valuable discoveries made during this period, of which mention may be made of bacteriolysis by Pfeiffer, in 1894; of bacterial agglutinins by Gruber and Durham, in 1896, and first utilized in the diagnosis of typhoid fever by Widal and Grünbaum, in 1896; of bacterial precipitins by Kraus, in 1897, and of blood precipitins by Nuttall, in 1899; of opsonins by Denys and Leclef, in 1895, but especially studied by Wright, Neufeld, Hektoen and others, in 1904, and finally capped by the brilliant discovery of the phenomenon of hemolysis by Bordet, in 1898, and in the same year of complement-fixation by Bordet and Gengou, which, in 1906, culminated in the application of its principles in the serum diagnosis of syphilis by Wassermann, Neisser and Bruck and independently by Detre, and constituting the most important single serum diagnostic test known at present to medical science.

These discoveries, unparalleled in brilliance and importance, naturally resulted not only in opening up a wide and useful field in the serum diagnosis of disease, but especially and more importantly a rich field in vaccine and serum therapy for the prevention and treatment of disease. Mention need be made of but such few examples as typhoid vaccination by Sir Almroth Wright, the father of modern vaccine therapy; the antitoxins for diphtheria and tetanus, already mentioned; antipneumococcus serum by the Klemperers, in 1891; antistreptococcus serum by Marmorek, in 1895; antianthrax serum by Sclavo, in 1901; antimeningococcus serum by Jochmann, Park, Flexner, Kolle and Wassermann, in 1905, as well as in more recent years the production of antidysentery, antigangrene, antiscarlet fever, antierysipelas and other sera. When Schick, in 1913, discovered his intracutaneous test for immunity in diphtheria, followed in the same year by von Behring's discovery of vaccination against this disease by toxin-antitoxin mixtures, the way was opened for the eradication of this dreadful scourge of childhood. Furthermore, the trail was blazed for similar results in scarlet fever, following the discovery by the Dicks, in 1923, that this disease was caused by a streptococcus, which almost every bacteriologist had seen in cultures of the throat of the disease since the early eighties, but which required the persistence of its discoverers, along with the bravery of five volunteers who submitted to inoculations with its toxin, to prove the etiological relationship. Almost at once a skin-test

for immunity was evolved, along with a method of vaccination and the production of an antiserum, which are proving useful in the prevention and treatment of the disease.

When, in 1896, the little son of Professor Langerhans died almost immediately after an injection of diphtheria antitoxin, serum therapy received an immediate check; but it was not until 1898 that Richet threw light upon the phenomenon, coined the word, "anaphylaxis," and opened up the way for the investigations of Arthus, Theobald Smith, von Pirquet, Rosenau and Anderson and others upon this new immunological riddle, which Besredka so aptly stated left the rules of immunity "standing on their heads." But, thanks to these pioneer investigators, followed later by those of Meltzer, Wolf-Eisner, Walker, Doerr, Weil and others too numerous to mention, the phenomena of hypersensitiveness are much better understood and its wide ramifications in clinical medicine, especially in relation to hayfever and bronchial asthma, thoroughly appreciated, along with the evolvement of improved methods for diagnosis by skin-tests and encouraging results in prevention and treatment.

Needless to state, chemistry in the meantime was not being neglected, and especially since, fifty years ago, Carl Ludwig had already stimulated great interest in it by his labors and discoveries, followed by those of Hoppe-Seyler, Salkowski and a veritable army of others too numerous to mention at the present time, but resulting in the widespread use of chemical analyses of the blood, urine, gastric and other secretions in the diagnosis and treatment of disease. But I believe special mention should be made of chemotherapy as the newest department in chemistry and therapeutic science, which had its origin largely in the work of Ehrlich, who as a medical student was greatly impressed with Heubel's work on lead-poisoning. This gave him the idea "that the ways and means by which drugs are distributed over the body must be of the greatest importance in the rational development of therapeutics." This thought guided him into a valuable series of studies upon the selective affinity of synthetic dyes for blood and other body cells and these in turn quite naturally into a consideration of the selective affinities of chemical agents for parasites, which led him to pronounce *corpora non ajunt nisi fixata* as a basic principle for this new science—that parasites can only be destroyed by those materials for which they have a certain affinity and by which they are fixed or "anchored" by the parasites. This combining affinity of para-

sites for chemical agents was designated by Ehrlich as *parasitotropy*, but he also knew that they had a combining affinity for the cells of the body designated as *organotropy*, and that only those substances can be employed as medicaments in which organotropy and parasitotropy stand in proper proportions.

Doubtless, the discovery of natural and acquired antibodies in diseases, and especially the protection and curative activity of diphtheria antitoxin, greatly strengthened Ehrlich's hope and dream of evolving chemical agents capable of reaching and destroying the parasitic causes of disease without injury to the cells of the host. As he stated on one occasion, the antibodies have "no affinity at all for the substances of the body, but they rush exclusively at the parasites. Therefore, in this case organotropy is reduced to zero, parasitotropy is absolute and the antibodies represent magic balls which seek their unerring aim." He never hoped that chemotherapy would ever reach this ideal of specific serum therapy, but that with chemical agents "we shall be obliged to use all our strength to take our aim very accurately in order to hit the parasites as much as possible, the body as little as possible."

But the hopes for serum therapy of various bacterial diseases aroused by the remarkable success of diphtheria and tetanus antitoxins, did not fully materialize, and when it was realized that for certain forms of infection, several of which had proved to be infections by protozoa, the body was unable to produce antibodies sufficient for eradicating the disease and that curative immune serums could not be produced, the question arose whether, with the rapidly growing field of synthetic chemistry, man could not match Nature's achievements by synthesizing chemical compounds specifically adapted for uniting with and killing the parasites when administered in subtoxic amounts.

Already, in 1887, synthetic chemistry had evolved acetanilid (antifebrin) and other substances from coal-tar, and while these antipyretics, narcotics and anesthetics were but symptomatic remedies, yet their production opened up a wide field for investigation in chemotherapy. Naturally the synthetic coal-tar dyes first attracted the attention of Ehrlich and his colleagues, and when Laveran and Mesnil showed how trypanosomiasis could be produced experimentally in mice and rats, the conditions were ripe for therapeutic experimentation in this field, which soon culminated in the discovery by Ehrlich and Shiga of a trypanocidal dye designated

as "trypan-red." This was soon followed by the discovery of "trypan-blue" and "Afridol violet" by Mesnil and Nicolle, the latter compound being of special significance because it differed from any previously tested in that its central nucleus was diamino-diphenyl-urea and the forerunner of that valuable colorless, water-soluble salt of a complex sulphonic acid synthesized fifteen years later and known as "Bayer 205," for the treatment of trypanosomiasis.

In the meantime, the parasite of syphilis having been discovered by Schaudinn, and the trypanocidal activity of atoxyl, a drug which had been synthesized in the late sixties by an obscure French chemist, Beauchamj, having been discovered by Thomas and Breinl, Ehrlich began his epoch-making work with the organic arsenicals which so impressed him with chemotherapeutic possibilities that he was led to announce: *Quod non sanat ferrum, sanat arsenicus; quod non sanat arsenicus, sanat ignis* (What iron does not cure, arsenic cures; what arsenic does not cure, fire cures) and ending with a complete justification of his faith by the synthesis of salvarsan (arsphenamin), the greatest triumph of modern chemotherapy.

Thus it will be seen that modern chemotherapeutic science is based upon the work and discoveries of many individuals, not only in the realm of synthetic chemistry, but likewise in the fields of parasitology and immunology. No doubt the discovery of the trypanosomes and spirochetes and the means of producing experimental infections on a large and cheap scale was a wonderfully fortunate circumstance, for had the early experiments in chemotherapy been conducted with the bacteria, disappointments would probably have extinguished enthusiasm by a lack of sufficient success and encouragement as subsequent events have amply indicated. To this extent "chance" surely played an important part, just as it did when the discovery of *Spirocheta pallida* induced Wassermann and his colleagues and Detre to try the complement-fixation test in syphilis, in which disease experience has since taught us the phenomenon is more marked and successful than in any other infection.

It is true that some of the theories of Ehrlich are no longer tenable in the light of more recent discoveries, and that the underlying principles of chemotherapy are far more complex than he originally conceived them, but yet it is to be remembered that they served him and his colleagues well and proved a wonderful stimulus to

chemotherapeutic research, just as his "side-chain" theory of immunity stimulated research in the field of immunology. In his opinion, scientific imagination and a healthy optimism were essential for success, and whoever had the opportunity of coming into close contact with him at the time when he worked in small and modest laboratories with few assistants was impressed by the fact that his main activity lay in the mental penetration of problems, as the lack of means permitted him to employ only the simplest technic. But he soon realized that chemotherapeutic discoveries would require a vast amount of work and ample resources. His accomplishments in the field of chemotherapy, which in a certain degree always will be "the sport of experimentation and of chance," were not entirely due to accidental success, but largely the outcome of prolonged, laborious and well-systematized laboratory experiments, constituting the "powder and dynamite" of scientific research, and the labors of which can be only fully appreciated by those engaged in similar work.

Thanks to Ehrlich's labors, chemotherapy has already accomplished a great deal in the treatment of numerous protozoan and metazoan diseases. Even in the far more difficult field of bacterial diseases, results have been so encouraging that the late Morgenroth has written: "What yesterday seemed absurd, tomorrow will seem banal. Today it is no longer a question for debate whether or not there can be a prophylactic and curative chemotherapeutic antisepsis, but merely a question of when, where and how. The future of chemotherapeutic antisepsis will probably resemble the present situation in respect to local and general anesthesia, for the future surgeon will inquire in each case whether the antisepsis shall be local or be general through the circulation, which antiseptic is to be selected, what concentration and quantity, etc., just as he now does with his anesthetics."

Naturally, with the discovery of the pathogenic bacteria and of chemical germicides, great hopes were entertained in the early eighties that the treatment of bacterial diseases, and especially those of suppuration, was finally solved. Experience soon showed, however, that to kill bacteria in the tissues was entirely different from their destruction in the test-tube, although Lister had already demonstrated the greatest single blessing in surgery, namely, antisepsis by phenol and the chlorides of mercury and zinc. But the method was soon superseded by that of aseptic surgery, only

to be revived again during the recent World War by Carrel and Dakin in the treatment of suppuration, the former discovering the correct principles underlying the successful chemotherapy of local infections by topical application. Who can deny the possibilities and probabilities of successful chemotherapy in tuberculosis and other systemic bacterial infections if money and men are available? And who can estimate even approximately the significance and benefits to humanity of such discoveries which we have every right to expect in this new science of chemotherapy?

In conclusion, I could wish for more space for reference to numerous discoveries in physiology during the past fifty years, but mention can be made of but one among the more recent, namely, that of insulin by Banting and his colleagues a few years ago. When one realizes its lifesaving properties and its great boon to countless diabetics, one must feel that the finger of God has rested upon its young discoverer, a would-be orthopedic surgeon, and that to be a discoverer in medicine with the possibility of helping suffering humanity is indeed a special blessing of Providence and a priceless privilege.

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1926

MR. PRESIDENT: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1926:

Total number of volumes in the Library, including the bound volumes and 21,823 unbound "Reports" and "Transactions"	149,739
Number of unbound "Theses" and "Dissertations"	26,397
Number of unbound Pamphlets	163,786

Included in the above are 4305 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2864 volumes more or less incomplete.

The duplicates, which number 2509 at this date, are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	107,089	2,854	109,943
Lewis Library	13,798	44	13,842
On permanent deposit:			
S. D. Gross Library	3,721	3	3,724
Library of the Obstetrical Society of Philadelphia	217	0	217
Mütter Museum*	190	0	190
			127,916

Received during the year from all sources, 3529 volumes, 15,628 pamphlets and 16,284 numbers of various periodicals.

* The books of the Mütter Museum are not permitted to be removed from the College building.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	2,882	15,533	15,686
Lewis Library	11		
S. D. Gross Library	3		
By purchase, General Account	330		
In exchange	303	95	598
	3,529	15,628	16,284

Accessions (including 57 volumes of "Reserves"), 2417.

General Library	2,403
Lewis Library	11
S. D. Gross Library	3
	2,417

Total increase in the number of volumes for the year: 2417.

Photographs received in response to requests sent out during the past year and voluntary gifts: Fellows of the College, 7; other than Fellows, 8.

Total number of portraits listed, 12,375.

The individual "Donors" for the year ending November 1, 1926, number 404; this represents 924 distinct presentations. Each gift is duly acknowledged.

The following list shows individual gifts of twenty volumes and over; also the number of volumes presented by the various publishing houses:

	Volumes.
Dr. Edward W. Watson	390
Dr. J. Norman Henry	101
Dr. S. W. Moorhead	96
Presbyterian Historical Society	94
Dr. Alfred Stengel	69
Dr. George M. Piersol	63
Mrs. S. Lewis Ziegler	49
Dr. Maurice Ostheimer	45
Dr. John H. W. Rhein	44
Dr. Joseph Walsh	41
Dr. Burton Chance	24
Dr. Alfred Hand	22
Dr. Evarista A. Calves	21
Dr. A. O. Allis	20

From the publishing houses of:

	Volumes.
P. Blakiston's Son & Co.	24
F. A. Davis Company	16
Lea and Febiger	23
J. B. Lippincott Company	18
W. B. Saunders Company	43

The Library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. H. B. Allyn
Dr. A. P. C. Ashhurst
P. Blakiston's Son & Co.
Dr. Charles W. Burr
Dr. A. A. Cairns
Dr. Burton Chance
Dr. Francis X. Dercum
Dr. W. A. Newman Dorland
Dr. A. A. Eshner
Dr. Thomas H. Fenton
Dr. J. C. Gittings
Dr. S. McC. Hamill
Dr. Alfred Hand
Dr. Hobart A. Hare
Dr. W. W. Keen
Dr. John A. Kolmer
Dr. Edward B. Krumbhaar
League of Nations
J. B. Lippincott Company
Dr. Edward Martin
Dr. Charles K. Mills
Dr. Elliston J. Morris
Dr. B. B. Neubauer
Dr. Maurice Ostheimer
Henry Phipps Institute
Dr. George M. Piersol
Dr. John H. W. Rhein
W. B. Saunders Company
Dr. George E. de Schweinitz
Dr. Alfred Stengel
United States Public Health Service

756 new publications were added to the Library during the past year, viz.: United States, 319; Great Britain and Colonies, 254; France, 59; Germany, 102; Italy, 4; all other countries, 18. Of these new publications, 37 were written or edited by Fellows of the College.

25 volumes were presented by the following authors or editors:

Dr. Maude E. Abbott
Dr. Lewellys F. Barker
Dr. Benjamin N. Bogue
Dr. Henry H. Donaldson
Dr. Lawrence F. Flick
Dr. Coleman R. Griffith
Dr. Hobart A. Hare (Editor)
Dr. Chevalier Jackson
Dr. W. W. Keen
Dr. Samuel W. Kelley
Dr. Alymer Maude
Dr. Francis R. Packard
Dr. Lewis S. Pilcher
Dr. Charles F. Prentice
Dr. William Allen Pusey
Dr. David Riesman
Dr. Nathan Rosewater
Dr. C. Howard Searle
Dr. Harry C. Solomon
Dr. William P. Vail
Dr. James J. Walsh
Dr. George H. Weaver

9 volumes were sent by the publishers at the request of the following authors or editors:

Dr. William Browning
Dr. Kedarnath Das
Miss Mary L. Hicks
Dr. Eldridge L. Eliason
Dr. Edward Jackson
Dr. Thomas McCrae
Dr. Charles C. Miller

Summary of the "Funds:"

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	49	$171.93
Luther S. Bent Fund	26	45.19
William T. Carter Fund	4	17.54
Gerardus Clarkson Fund	20	58.30
Francis X. Dercum Fund	49	236.34
Louis A. Duhring Fund	47	377.55
John D. Griscom Fund	104	224.85
Charles T. Hunter Fund	2	5.92
William F. Jenks Fund	77	486.62
Oliver A. Judson Fund	10	54.74
William V. and John M. Keating Fund	16	33.07
William W. Keen Fund	46	99.70
Library Endowment Fund	130	655.35
Horace Magee Memorial Fund	134	521.52
J. Ewing Mears Fund	35	156.60
Charles K. Mills Fund	6	23.04
Weir Mitchell Fund	58	175.98
E. E. Montgomery Fund	17	81.13
John H. Musser Fund	9	25.33
Elizabeth K. Newcomet Fund	14	29.66
William F. Norris Fund	38	199.43
Charles A. Oliver Fund	18	57.39
Philadelphia Medical Society Fund	20	86.51
Lewis Rodman Fund	43	211.99
Douglas Stockton Warren Fund	36	88.63
John F. Weightman Fund	27	20.67
J. William White Fund	24	113.58
Caspar Wistar Fund	50	195.49
	1,109	$4,454.05

Special accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	175	$150.11
Fund for rare and valuable books	27	865.70
S. D. Gross Library Account	1	43.02

	Expended.
Catalogue Endowment Fund—on account of salaries	$46.50
Louis A. Duhring Fund—general purposes of the Library	11,500.00
Morris Longstreth Library Fund—on account of salaries	965.76
George B. Wood Fund—Library supplies, stationery, etc.	309.56

Number of volumes bound, 1326.

	Visitors.	Fellows.	Total.
Use of the Library	5,179	1,588	6,767

The Library has been kept open two evenings each week and on the six minor legal holidays for the same period of time as during the previous year:

Evenings.	Visitors.	Fellows.	Total.
75	739	171	910

Legal holidays.	Visitors	Fellows.	Total.
6	61	19	80

These figures are included in the total given for the year.

	1926.	1925.	Increase
Number of volumes consulted in the Library	29,862	23,512	6,350

The number of books "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes and works of reference kept on the shelves in the Reading Room and the Fellows of the College have access to the Book Stacks. There are, therefore, a large number of volumes consulted of which no accurate record can be kept.

	1926.	1925.	Decrease.
Number of volumes taken out:	3,559	4,606	1,047

Use of Study Rooms: Average number of rooms in use throughout the year, 5. Greatest number in use at one time, 10. Daily average of the number of volumes in use in these rooms, 149.

	Works.	Volumes.	Cards written.	Printed cards revised.
Cataloguing	887	899	3,964	2,340

All books added to the shelves during the past year and 104 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 7983 cards have been revised, rewritten and filed.

3333 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject and by author under the subject. The binding of the pamphlets by subjects will be started in the coming year.

We have listed November 1, 1926, 1216 current periodical publications, including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Funds	82	307
By purchase from General Account	42	193
In exchange	51	132
Editors	83	31
Publishers	39	10
Through the Courtesy of the Editor of the *Therapeutic Gazette*, current numbers are received at stated intervals	40	48
	337	721
Reports, not included in the above	141	17
	478	738

The actual number of periodicals received during the year, including new subscriptions (2 American, 7 foreign) total 1058.

The following is a list of the foreign schools of medicine with which we exchange publications at the present time:

University of Amsterdam
" Basel
" Berlin
" Bonn
" Breslau
" Erlangen
" Geneva
" Giessen
" Göttingen
" Halle
" Heidelberg
" Kiel
Faculty of Medicine of Paris
University of Königsberg
" Lausanne
" Leiden
" Leipzig
" Liège
" Lund
" Marburg
" Rostock
" Strassburg
" Upsala
" Utrecht
" Würzburg
Faculty of Medicine of Toulouse

During the past year we have heard from Amsterdam, Basel, Berlin, Breslau, Erlangen, Giessen, Göttingen, Halle, Kiel, Königsberg, Lausanne, Leipzig, Liège, Marburg, Paris, Upsala and Utrecht. 1173 dissertations and 831 theses have been added to our collection.

We have received in cash from the sale of duplicates for the current year ending November 1, 1926, $287.85.

We have distributed books and journals on exchange account to the following:

Boston Medical Library
League of Nations Medical Library, Geneva
Medical Library, McGill University
Medical Society, County of Kings, Brooklyn
New York Academy of Medicine

We have received exchanges from:

Boston Medical Library
Columbia University, College of Physicians and Surgeons
League of Nations Medical Library, Geneva
Medical Library Association
Medical Library, McGill University
Medical Society, County of Kings, Brooklyn

With the aid received from exchanges and purchases made with funds appropriated for the purpose by the Library Committee, we have been able to complete the files of the following journals:

Annali di Odontologia, Roma
Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, Jena
Revue Illustrée de Polytechnique Médicale et Chirurgicale, Paris

Amount of fines collected for the current year ending November 1, 1926, $100.65.

List of rare medical books and works of special interest received during the year (these rare books have been acquired without in any way interfering with the purchase of current medical publications):

INCUNABULA

(Total number of incunabula at this date, 371 Titles, 355 Volumes.)

Alexander Aphrodisaeus s. Aphrodisiensis. Problemata, lat. per Georgium Vallam. Venetiis, Antonius de Strata, Cremonensis, 1488. (Hain 658.)

Fund for Rare Books.

Buch der Natur.*** Cunrat von Megenberg von lateyn in teutsch transsferieret und geschriben nat.*** Augustae Vindelicorum, Johannes Baemler, Augustensis, 1475. [Hain 4041.]
[*27 ff. supplied in fac-sim.*]
[*All plates supplied in fac-sim.*]

Fund for Rare Books.

Burlaeus s. Burley, Gualterus. [Expositio super octo libros physicorum.] Venetiis, Octavianus Scotus, Modoetiensis, per Bonetum Locatellum, Presbyter Bergomensis, 1491. [Hain 4135.]

Fund for Rare Books.

Diodorus Siculus. [Bibliotheca seu historiarum priscarum libb. VI, e graeco in latinum traducti per Fr. Poggium.] Venetiis, Guillielmus [de Plano] Cereto de Tridino de Monteferrato cognomento anima mia, 1496. [Hain *6191.]

Fund for Rare Books.

Works of Special Interest

Albinus, Bernhard Siegfried. Anatomy of painting; or, A short and easy introduction to anatomy*** to which are added the anatomy of Celsus, with notes, and the physiology of Cicero. With an introduction*** by John Brisbane. London, Scott, 1769.

In Exchange.

Alpinus, Prosper. Presages of life and death in diseases. Translated from the last Leyden ed. revised and published by [H. D.] Gaubius, at the request of Hermann Boerhaave, by R[obert] James. London, Strahan, 1746.

Fund for Rare Books.

Andree, John. Considerations on bilious diseases, and some particular affections of the liver, and the gall bladder. 2 Ed. London, Murray, 1790.

Presented by The Pennsylvania Hospital.

Arnould, R. P. F. Traictez curieux, de medecine et chirurgie. Le premier contenant une doctrine generale, des fractures du crane, reduitte en forme d'abregé par un sçauant homme. Le second est un tres excellent traicté de la dissenterie, composé par Guillaume Fabri, medecin de Hilden. Le troiséme les reuelations charitables, de plusieurs remedes souuerains, contre les plus cruelles et perilleuses maladies, qui puissent arriuer au corps humain. Rouen, Chez François Vavltier sous la porte du Palais, prés Bastille, 1661.

William W. Keen Fund.

Arrigoni, Antonio. Della mania, della frenesia, e della rabbia. Dissertazione. Milano, Nella stamperia di Antonio Agnelli, 1757.
Francis X. Dercum Fund.

Barletti, Caro. Analisi d'un nuovo fenomeno del fulmine ed osservazioni sopra gli usi medici della elettricità. Pavia, Nella stamperia del R., ed I. Monistero di S. Salvatore per Giuseppe Bianchi con permissione, 1780.
Francis X. Dercum Fund.

van Beughem, Cornelius. Incunabula typographiae.*** Amsterdam, Wolters, 1688.
[*The earliest known separately printed catalogue of incunabula.*]
Fund for Rare Books.

Bonet, Théophile. Medicina septentrionalis collatitia, sive rei medicae, nuperis annis à medicis Anglis, Germanis et Danis emissae, sylloge et syntaxis. Volume 1. Genevae, Chovët, 1684.
Henrietta Rush Fales Baker Fund.

Budaeus, Gottlieb. Miscellanea medico-chirurgica, practica et forensia.*** Leipzig, Joh. Gottlob Laurentius, 1732.
Fund for Rare Books.

Coulet, Etienne. Le gouteux en belle-humeur; ou, L'Eloge de la goute*** mis au jour par [Nicholas] de Gueudeville. La Haye, Aux depens de la compagnie, 1743.
In Exchange.

Dease, William. Observations on the different methods of treating the venereal disease. Dublin, Williams, 1779.
Presented by the Pennsylvania Hospital.

Deschamps, Abbé. De la maniere de suppléer aux oreilles par les yeux.*** Paris, Debure, 1783.
Fund for Rare Books.

Dieterich, Joh. Cunradus. Iatrem Hippocraticum; continens narthecium medicinae veteris et novae*** juxta ductum aphorismorum Hippocratis.*** Ulmae, Kühne, 1661.
[*Greek and Latin text.*]
Fund for Rare Books.

Ersfeldius, Nicolaus Wilhelmus. Dissertatio inauguralis medica de rhachitide.*** Jena, tip. Viduae Samuelis Krebsii, 1682.
Fund for Rare Books.

Fabricius Hildanus, Guilhelmus. Lithotomia vesicae; that is an accurate description of the stone in the bladder.** Done in English by N. C. [Nicholas Culpeper.] London, Norton, 1640.
Fund for Rare Books.

Freytag, Gottlieb. De Sanguine, ejusque observatione, quas contra vulgarem de quibusdam opinionem.*** Altdorphi, e typo. Academico Hageniano, 1660.

Fund for Rare Books.

Galart de Montjoye. Sur le magnétisme animal, où l'on examine la conformité des opinions des peuples anciens et modernes.*** Paris, Duplain, 1784.

Luther S. Bent Fund.

Guillemeau, Jacques. Oevvres de chirurgie*** avec les portraits et figures de toutes les parties du corps humain et des instrumens necessaires au chirurgien. Paris, Marchant, 1598.

Fund for Rare Books.

Guy de Chauliac. La grande chirurgie.*** Restituée par Laurens Joubert. Rouen, David du Petit Val, 1615.

Fund for Rare Books.

Henderson, William. A few observations concerning those things which are probable, or in some measure ascertained, relative to the history and cure of the plague. London, Murray, 1689 [1789.]

Presented by The Pennsylvania Hospital.

Hippocrates. Aphorismorum Hippocratis carmine redditi jamque deno editi a Joanne Lvdovico Gansio, franco poëta L. C. Argentorati, Lederatz, 1624.

Fund for Rare Books.

Hoffmann, Joh. Mauric. Disputatio inauguralis medica de paroxysmis frequentioribus, scil. febrilibus, asthmaticis, epilepticis, colicis, et hystericis. Altdorfi, Meyeri, 1675.

Fund for Rare Books.

Hollerius Stempanus, Jacobus. De morbis internis libri II illustrati.*** Ludovici Dureti*** et Antonii Valetii.*** Francofurdi, Wecheli, 1591.

William W. Keen Fund.

Hortus sanitatis. Herbarius zu teütsche.*** Mainz, Schöffer, 1485.
[*fac-sim. München, Mandruck, 1925.*]

By Purchase.

Jurin, James. An abstract of the case of James Jurin, M.D.*** As far as relates to the taking of his lixiuium for the stone and gravel. [London] 1767.

Fund for Rare Books.

Kerckringius, Theodorus. Anthropogeniae ichnographia sive conformatio foetus ab ovo usque ad ossificationis principia in supplementum osteogeniae foetuum. Amstelodami, Sumptibus Andreae Frisii, 1671.
William F. Jenks Fund.

Klug, Daniel Godofredus. De fluxu chyli in fluore muliebri gonorrhoea coeliaca urinis, lacteis et lactis abundantia thematis novi summaria et theses. Altdorfi, typ. Henrici Meyeri, 1675.
Fund for Rare Books.

Lactus, Joh. Ciarlatanaria medicorum. Oder Marcktschreyerey der gelehrten Aerzte mit Fleiss in verständlich Teutsch gesetzt. Freysingen, auf Kosten guter Freunde, 1717.
Fund for Rare Books.

Ludekens, Esaias. Theses medicae de transitu sanguinis per medium septum impossibili. Altdorphi, Hagen, 1659.
Fund for Rare Books.

Macer Floridus, Aemilius. De herbarum virtutibus*** cum*** Georgii Pictorii*** expositione, antea nunquam in lucem edita.*** Basilieae, Petri, 1559.
Horace Magee Memorial Fund.

Maynwaring, Everard. Medicus absolutus. ΑΑΣΣΗΟΙΟΣ. The compleat physitian, qualified and dignified.*** London, printed for the booksellers, 1668.
Gerardus Clarkson Fund.

[Newton, Sir Isaac.] Optics; or, A treatise of the reflexions, refractions, inflexions and colours of light. Also two treatises of the species and magnitude of curvilinear figures. London, Smith, 1704.
Fund for Rare Books.

Piazzi, Matteo. Prattica per espurgare le case et robbe infette e sospette di contagio. Bologna, l'Herede del Benacci, 1630.
John D. Griscom Fund.

Pictorius Villinganus, Georgius. Sermonum convivialium apprime utilium, libri X, deque ebrietate lusus quidam.*** 3 Volumes in 1. Basileae, Petrina, 1571.
John D. Griscom Fund.

[Riolan, Jean.] Brevis excursus in Battologiam Quercetani quo alchymiae principia funditus diruuntur, et artis vanitas demonstratur. Parisiis, apud Hadrianum Perier, ex off. Plantiniana, 1604.
[*Rare first edition. Printer's device on title-page.*]
Fund for Rare Books.

Rustichelli, Pietro Torrigiano. Plus quam commentum in parvam Galeni artem cum duplici textus interpretatione, antiqua scilicet et Leoniceni, et eiusdem libello de hypostasi.*** Venetiis, apud Iuntas, 1557.
Fund for Rare Books.

Ryan, Michael. An enquiry into the nature, causes and cure of the consumption of the lungs; with some observations on a late publication on the same subject. Dublin, Gilbert, 1787.
Presented by The Pennsylvania Hospital.

Sachs, Philippus Jacobus. ΓΑΜΜΑΡΟΛΟΓΙΑ sive gammarorum, vulgo cancrorum consideratio physico-philologico-historico-medico-chymica.*** Francofurti, Fellgibelii, 1665.
[*Rare first edition of interesting work.*]
Fund for Rare Books.

Salernum, School of. De conservanda bona valetudine opusculum scholae Salernitanae.*** Antverpiae, apud Ioannem Bellerum, 1557.
Fund for Rare Books.

Saltzmann, Johannes Rudolphus. Kurtze beschreibung des heylsamen Badts und Bronnens der Sahlbronnen, oder das Sehlbacher Bad genant.*** Strassburg, Zetzner, 1612.
In Exchange.

Sanchez [Antonio Nuñez Ribeiro]. Historical investigation into the first appearance of the venereal disease in Europe.*** Translated from the French by Joseph Skinner. London, Johnson, 1790.
Presented by The Pennsylvania Hospital.

[Summer, Balthasar.] Artzneybüchlein sehr gut und nützlich einem jeden Haussvater täglich zu gebrauche.*** Wittenberg, Seuberlich, 1599.
Fund for Rare Books.

Sylvius, Jacobus. In Hippocratis elementa. Parisiis, apud Aegidium Corbinus, 1561.
Fund for Rare Books.

Tachenius, Otto. Tractatus de morborum principe, in quo plerorumque gravium ac sonticorum, praeter naturam affectuum, dilucida enodatio, et hermetica, id est, vera et solida eorundem curatio proponitur.*** Osnabrugi, Schwänder, 1678.
Fund for Rare Books.

Thriverus Brachelius, Hieremias. In Hippocratem De ratione victus in morbis acutis. Lvgdvni, apud Haeredes Iacobi Iuntae, 1552.
Horace Magee Memorial Fund.

Tickell, William. Concise account of a new chymical medicine, entitled, Spiritus aethereus anodynus, or, anodyne aethereal spirit.*** Bath, Salmon, 1787.

Fund for Rare Books.

Manuscripts

Stillé, Alfred. Truth in medicine and truth in life inseparable. (Phila., 1885.)

Presented by Edward H. Goodman, M.D.

Other Interesting Additions

Stethoscope belonging to the late Dr. Willard Parker.

Presented by Harlow Brooks, M.D.

Two holograph letters of Emil du Bois-Reymond; a holograph letter of Carl Ludwig and postal card photographs of Ewelme Church and Ewelme, St. John's Hospital with an autograph letter of I. A. Dodd, showing that the Regius Professor of Medicine at Oxford is Master of the Ewelme Almshouse and receives a salary from the endowments.

Presented by Albert P. Brubaker, M.D.

Microscopical preparations and photomicrographs of tissue used by Dr. Thomas Hodgkin in describing the disease that goes by his name; also a note book of Dr. William Darrach, 1796–1865, and a Poster for the Cholera Epidemic of 1832 prepared by him.

Presented by Herbert Fox, M.D.

Photostat negative of the "Virginia Acts 1736–1794" relating to medicine.

Presented by W. W. Keen, M.D.

"Snap Shots of Places of Medical Interest in Newport, R. I.;" "A Short Sketch of the Redwood Library and Athenaeum;" King's "Historical Sketch of the Redwood Library and Athenaeum;" and a clipping from "The Providence Sunday Journal, June 21, 1925."

Presented by William S. Sherman, M.D.,
through the courtesy of W. W. Keen, M.D.

A metal plaque of Lister, a reproduction from the original plaque in the University of Rochester.

Presented by Snead & Co.

Photographs of two portraits of Galen.

Presented by Joseph Walsh, M.D.

1. Antique Sinhalese Ola (c. 1650 A.D.).
2. Sixteenth Century Monastic Stylus for writing on prepared fronds of Talipot and Palmyra Palms.
3. Ceremonial Dagger and Stylus of a Sinhalese Physician (c. 1550).

Presented by Casey A. Wood, M.D.
through the courtesy of George E. de Schweinitz, M.D.

We have also received during the past year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 359 prints, and to others than Fellows, on application, 883 prints. These outside applications include requests from Baltimore; Chicago; Denver; New York; Ontario, Canada; Pittsburgh; Philadelphia, and San Marino, California.

The work for the Library has been continued as heretofore according to the instructions of the Library Committee; 2715 large and small prints have been made; 15 volumes of incunabula completed as well as various portraits and manuscripts. Up to the present time there have been made photostatic reproductions of 240 incunabula.

There has been an increase to the Library Endowment Funds as follows during the past year: Louis A. Duhring Fund, $3334.39; Richard H. Harte Fund, $5000; Charles T. Hunter Fund, $489.73; Fund for Binding, $420; E. E. Montgomery Fund $1000; J. William White Fund, $5000; and the George B. Wood Fund, $50. A complete list of all the Funds and the expenditures for the year is given in a previous part of this report. All additional expenses, salaries, binding, etc., have to be governed according to the funds appropriated by the College to the Library Committee annually.

During the past summer a seventh tier was added to the Book Stacks; the cost of this, about $15,000, was defrayed from funds accumulated for the purpose for a number of years by the Library Committee. This seventh tier will accommodate about 50,000 volumes, and will permit, according to the average of the last ten years accessions, 3000 volumes per annum, of about sixteen years' growth. The stacks are built on concrete piles and there can be additional vertical enlargements by removing the roof. If such is determined upon in the future, then arrangement should be made for an automatic electric passenger elevator in the stacks.

This seventh tier of stacks, on account of extreme heat and other minor obstructions, was not completed until September 3, leaving the rearrangement of the entire contents of the Book Stacks to be made after the Library was open to readers. The books have been rearranged and the periodicals will be arranged as opportunity permits without interfering with the use of the Library.

The figures in the present report show that the affairs of the Library have progressed in a satisfactory manner; in the number of new publications received, as well as the receipt of a number of valuable curios; an increase to three funds, Richard H. Harte Fund, $5000; Charles T. Hunter Fund, $489.73, and the J. William White Fund, $5000; to the Library Endowments; a new seventh tier of stacks and the rearrangement of the contents of nearly half of the entire stacks. To a certain extent the work has been retarded from the fact that there have been ten changes in the minor library force during the current year, and each new employee must be instructed in the workings of the library before being capable of attending to any duties in a practical manner.

There have been an unusual number of visitors from all parts of this country and abroad; the appointments of the library received unstinted praise; the arrangement of the current periodicals in the Norris Room was especially commented upon; while some had seen a somewhat similar arrangement on a smaller scale, not one had seen a display of what might be considered the current medical periodical literature of the world in such an accessible arrangement.

The photostat reproductions of incunabula were a revelation to all; the work was applauded and the fact that access to these rare and in some cases priceless works was free to the research workers of the world was highly commended.

Library Committee,

Francis X. Dercum, M.D.,
Chairman.

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